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SYMPOSIUM.

THE NEURAL MECHANISM OF HEARING.

V.—ETIOLOGICAL AND CLINICAL TYPES OF SO-CALLED "NERVE DEAFNESS."

(a)—"NERVE DEAFNESS" OF LITTLE OR UNKNOWN PATHOLOGY OR ETIOLOGY.

VARIATIONS IN VERTIGO AND TINNITUS WITH DEAFNESS. MENIERE'S SYMPTOM COMPLEX.

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The term Méniére's disease is a misnomer. There is no distinct separate pathological entity which gives rise to paroxysmal vertigo, tinnitus and deafness. For convenience, when these symptoms appear without some clear pathological cause, such as labyrinthitis, the condition has been called Méniére's disease.

A large number of cases present at times the symptoms of vestibular irritation. Our first problem is to select the cases which may be classified properly as Méniére's. This is done for the most part by exclusion.

Cases of toxic auditory neuritis caused by the poison of infective foci, of syphilis, must not be included in this category. We should not classify a case as a Méniére's until

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a careful neurological and laboratory checkup has failed to reveal pathological disorders sufficient to cause the triad of symptoms under consideration.

Unfortunately for the sake of simplicity, all of these Ménière cases are not cut from the same pattern. Some are caused by Eustachian tube obstruction, others are caused by increased intralabyrinthine pressure from within the cranium; some are caused by an excess of water or of sodium chloride in the labyrinth, while others are caused by physical changes in or around the vestibular branch of the auditory nerve. We shall later consider these possibilities *seriatim*. When we have decided that the case is one which has Ménière's symptom complex, our next duty is to ascertain the etiological factor responsible for the syndrome.

CONSTITUENTS OF MENIERE'S SYMPTOM COMPLEX.

Deafness: This is sometimes the first symptom to affect the patient. It is generally unilateral. I have found invariably diminished bone conduction, with a dip in both the lower and the upper limits. The upper limit is particularly affected. The deafness is usually slowly progressive.

Tinnitus: This may be the symptom first noticed by the patient, although a hearing defect may have been present for years. Tinnitus is worse on one side. During an attack, the head noises may be much exaggerated in intensity. An aura or an unusual tingling feeling in the ear may precede the paroxysm.

Vertigo: This is sudden in onset. It is paroxysmal in character. The patient staggers and he is compelled to assume a recumbent position. The sufferer may fall and sustain a head injury. Nausea and vomiting are nearly always present. The vertigo is rotary in character, as if surrounding objects were moving around the patient. Nystagmus is commonly present during the attacks, sometimes to both sides. The paroxysms are few in number at first, but later become more numerous and are more severe. Dandy[1] says that the attacks often come during sleep and that the average duration is three to four hours. He calls attention to the protracted type of the vertigo as being very different from the dizziness that is induced by change of position.

Occasionally the patient becomes bedridden, because he is afraid of the vertiginous attacks. Oppenheim[2] describes a condition which he calls the status Méniére, in which dizziness and unsteadiness are constantly present between attacks.

The caloric test generally gives normal vestibular responses in these cases.

SOME CAUSES OF MENIERE'S SYNDROME AND TREATMENT.

Unilateral Eustachian Tube Obstruction: The English school of otologists stress this etiological factor more than we do.

Sydney Scott[3] thinks unilateral obstruction, with consequent inward pressure of the stapes on the labyrinth, the commonest cause of Méniére's syndrome. He noticed this first during the great war, in the airmen after high flying. He observed that when both drums were invaginated by the air pressure, deafness without vertigo was the result, but that when the drum retraction was unilateral, pronounced vertigo was prominent. Since then he has in many cases noted Méniére's symptom complex coincident with insufficiency of the Eustachian tube, and has proved this relationship by permanently relieving the symptoms when the patency of the tube was restored.

Muller[4] reports the association of nasal and postnasal catarrh with unilateral Eustachian tube obstruction in many Méniére's cases seen by him.

Tilley, when in this country a few years ago, expressed the same opinion and cited histories of typical Méniére's syndrome cases in which there was unilateral tubal obstruction and in which cure followed the removal of this cause.

Krassnig[5] reported 30 out of 32 patients with this syndrome as suffering from tubal obstruction. He thinks that an inflammatory condition of the tensor tympani muscle and of the stapedius prevents the regulation of the perilymph pressure.

The treatment for this Eustachian tube obstruction is obvious: catheterization and the use of the bougie, together with correction of any apparent nasal defect.

Faulty Water Metabolism and Excess of Sodium Chloride: In 1931, Dederding[6] reported three cases of Méniére's syndrome in connection with Quinke's circumscribed edema of the subcutaneous tissue. Dederding and Mygind[7] advanced the theory that Méniére's symptom complex is caused by an excess of water in the intralabyrinthine tissues increasing the tension. They reported success in treatment with a salt-free diet and a decreased intake of fluid.

Furstenberg[8] elaborated on this theory and advanced the idea that the excess of sodium in the body is the irritating factor. He reasoned that body water is chiefly a solution of sodium salts in water. He advocated a limitation of sodium in the body by diminishing the intake of sodium and through neutralization in the tissues by the administration of ammonium chloride. He has had marked success with these measures. In one case he was able at will to produce paroxysms of vertigo by the administration of sodium.

I have tried this treatment in several cases and have often found it efficacious. There are difficulties, of course. For instance, one patient was unable to take a small dose of ammonium chloride without diarrhea. Then, too, it is difficult to keep a patient for an indefinite period on a diet.

Abnormal Arterial Tension: Raised blood pressure is sometimes a cause of Méniére's syndrome. Atkinson[9] makes the point that the vertigo may be due to the pressure being insufficient for the patient's needs and advises digitalis and ergot.

An abnormally low blood pressure may also cause these attacks. Lake[10] first drew attention to this. Adrenalin, ergotine and cardiac tonics are indicated.

Abnormal Structural Changes: Dandy,[11] in a recent communication, proves the effect of direct pressure on the auditory nerve by large arterial branches or from adjacent tumors in producing typical Méniére attacks. These abnormalities were noted at operation. After nerve section, the Méniére attacks ceased.

Surgical Treatment: Dandy's series of operations has now reached 170 in 160 patients. His operation consists of section of the vestibular portion of the VIIIth nerve. His results have been eminently satisfactory.

Coleman and Lyerly,[12] in 1933, reported having operated in a similar way upon 10 patients, and report success. They do not operate unless the patients have been seriously incapacitated.

Cairns,[13] of London, and McKenzie,[14] of Toronto, also report a number of successful cases treated in this manner.

Portmann,[15] when in this country in 1927, advanced the theory that Méniére's syndrome is caused by pressure from the lateral cistern on the membranous labyrinth, through the saccus endolymphaticus. His operation consists of a section of the saccus. Dandy disagrees with this theory and apparently contraverts it.

In closing, I would say that during the past half-dozen years much has been done towards explaining the various causes of this complex. Some cases are comparatively easy to help, either by instrumentation, or by the aid of dietetic and therapeutic measures. Others, again, apparently are only aided by surgery. We can be grateful that the intensive work in this field has been fruitful in lighting up one of the dark corners in medicine.

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THE NEURAL MECHANISM OF HEARING.

V.—ETIOLOGICAL AND CLINICAL TYPES OF SO-CALLED "NERVE DEAFNESS."

FROM AFFECTIONS OF THE BRAIN (A) ORGANIC FACTORS.

CONGENITAL WORD-DEAFNESS.*

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The unsatisfactory term "word-deafness" has been used to describe a condition in which there is selective impairment of recognition of the spoken word, in contrast to other sounds. The acquired condition, arising from a lesion in the central nervous system, consists in the impairment of a previously efficient function. The congenital condition, which has not been identified with any pathological process, consists in a relatively slow and imperfect development of that function. Various observers have mentioned such a difficulty in children, and it has been described by Orton in this country, and Ewing in England.

The primary features of congenital word-deafness are a difficulty in the recognition of the spoken words of others, and a resultant speech disability—both of these conditions occurring in the presence of normal hearing at the points commonly tested on the auditory range, and in the presence of a potentially normal intellect. The syndrome of congenital or, as it is better called, developmental word-deafness, may be conveniently described under the four headings of audition, speech, other neurological aspects, and emotional aspects. The presenting complaint in children below the age of 6 years may be a question of imperfect hearing; delayed speech development; distorted speech; or any combination of these. Where the reduction in word recognition is moderate in degree, it may have escaped identification as such in the

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preschool years, so that a child of school age may exhibit such symptoms as auditory inattention, poor speech, difficulty with reading, or the school failure suggestive of general intellectual retardation.

Under the heading of audition, four conditions may be specified. There is, first, the imperfect understanding of the spoken language which the child has always heard used around him. This difficulty is more marked as the syllable and word sequences become more complicated, or less familiar in the child's experience. The response to any spoken words at all may be noted as very late—well after the second year of life—and contrasted with development in other respects. Next, the ability to discriminate meaningful sounds other than language—such as musical, mechanical, and animal sounds—is apparently intact. Practically speaking, these sounds constitute but a small part of the environment, yet a difference may be worked out in some cases between the recognition of these nonlanguage sounds, and the recognition of speech sounds and sequences. Third, and most important, the hearing is normal. In the cases on which these observations are directly founded, and some others, a Western Electric 2A audiometer was used to make the hearing tests. In other cases which seem to belong to this group, the estimation of hearing had to be made with informal, and less reliable tests, because of the immaturity of the child. Such cases, of course, should be subject to verification. After hearing, a fourth feature is auditory inattention, which is, logically, most prominent in regard to the spoken words of others.

Under the heading of speech, it may be said that this fraction of the language function characteristically appears late in these cases, and that it is both defective and scanty. Many of the defects are of the infantile type, with substitutions, elisions and certain unevolved consonants and their combinations as prominent features. In the sparse vocabulary are not only infantile word forms, but also frank neologisms which sometimes only remotely recall the exact form of the word from which they are derived. Since the lack of speech, or its imperfection, is a phenomenon in the motor sphere, it may be more certainly noted by parents and others; so that the speech, rather than a question of deafness, may frequently be first attended to.

There is no neurological evidence in these cases, either of a localized or of a progressive process. The chidren grow normally in the physical and in the physiological respects, except as noted. A ready visual attention is present, but it may be remarked that this is not directed at the lips of others, as is sometimes the case in untrained children with defective hearing. A generalized over-activity has been observed, and may be explained on the basis of a compensatory energy outlet. A delayed evolution of right- or left-handedness, or a mixed handedness pattern, has been noted, as well as a family history of mixed handedness and of other language disabilities. However, the number of cases of developmental word-deafness which have been studied in this way is too small, and the individual cases too various in age, intelligence and degree of difficulty to permit any conclusions as to the relationship of these factors. The various standardized psychometric tests are most important, in that they bring out the difference in capacity to perform in language and in nonlanguage situations. Where these measurements of intelligence are dependent on the immediate understanding of the spoken word, or on a previous experience in its comprehension, they are beyond the abilities of children of this type. Since the understanding of speech from the second year onward is an essential factor in the full development of the intelligence, the word-deaf child suffers a deprivation in this way, and his measurable intelligence is reduced in tests of the type mentioned. In other tests, however, where visual experience is the factor, a normal, or comparatively better, performance may be expected. The type of test situation must be taken into account in any attempt to estimate the underlying intellectual capacity of these children. In a child of school age, a specific reading disability is found as a consequence of lack of foundation in the recognition and use of the spoken language. This may be thought of as comparable to, although different from, the special reading situation in the cases of deaf children.

The emotional aspects of developmental word-deafness are of great importance. Abnormal behavior most frequently occurs, probably as a result of the constant exposure to situations in which the child is called upon to perform at a level beyond his capacities. The delayed and scanty speech has suggested to some that this condition is of emotional origin,

and that a conflict of some sort is finding its expression in a suppressed speech function. It is true that such emotional factors may greatly accentuate the negative trend in speech and other behavior; but the evidence, in the case of the word-deaf child as described, does not allow the conclusion that defective motivation is the difficulty in a failure to recognize spoken words, and a distortion of speech of this exact order.

The differential diagnosis in cases of this kind calls for the exclusion of a general intellectual defect and of regional deafness. A thorough history, thorough clinical observation and psychometric tests are the basis on which intelligence is to be judged. Allowance must always be made, as in the case of the deaf child, for the lack of certain responses, the development of which is dependent on an efficient recognition of spoken language. The suspicion of regional deafness can only be finally dismissed after a satisfactory audiometric rating.

There has been little opportunity for investigation of these cases over a long period, so that prognosis is uncertain. There is a tendency to slight spontaneous improvement with growth, and increased exposure to the language of others. Yet, this rate is so slow that a level of efficiency adequate to social adjustment and education is not to be reached. Actual experiments in the retraining of these children are still very limited, but experience in the comparable field of congenital word-blindness suggests that special teaching methods to stimulate the growth of this laggard function are eminently worth trying.

With no evidence of progressive pathology, and the possibility of development with growth, the condition is probably best regarded as a constitutional variant in this part of the language function. Such variants are more widely and more easily recognized in the cases of speech alone where there is good recognition of the spoken word and in written language, as manifested in reading and spelling confusions. Like these, the defect of word-deafness seems to be distributed in all degrees among the various afflicted individuals; and it may be connected, like these, with failure to establish a complete and efficient language function during the early years in the usual single cerebral hemisphere. In the meantime, its further investigation should be of interest to those concerned with the fields of otology, neurology and child development.

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FROM AFFECTIONS OF THE BRAIN (A) ORGANIC FACTORS.

ACQUIRED WORD-DEAFNESS.*†

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The term word-deafness is, as Dr. Orton has mentioned, a misnomer,[1] since the patient who is called word-deaf does not have any defect in hearing. He is not unable to hear words but has difficulty in understanding them. The subject is still further confused because of the fact that earlier writers considered word-deafness as a clearcut symptom which appeared as an isolated defect. A complete study of these earlier reports was recently made by Weisenberg and his coworkers.[2] They were unable to find a single case where a complete and adequate report of the language examination was given, and where word-deafness appeared as the only language defect. Head also reached the conclusion that word-deafness did not appear in the absence of other language disability.[3] My own experience, which supports this view, includes the examination of more than 300 patients with language disabilities. All of these patients were admitted to the Neurological Institute of New York because of a cerebral lesion. I did not encounter a single case of "pure word-deafness." Where word-deafness occurred, it was part of an aphasic state.

Language function is considered to be resident in only one cerebral hemisphere. It is, with a few exceptions, found in the hemisphere opposite to the preferred hand. Most work-

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†The patients were admitted to the Medical and Surgical Services of the Neurological Institute of New York. It was through the kindness of the Chiefs of Service that these studies were made possible.

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ers are in agreement that understanding the spoken word is dependent upon the convolutions of the temporal lobe of this dominant hemisphere.

Because of this general agreement upon the anatomical site, I have selected from my group of aphasics 17 who had verified brain tumors, primarily of this area. Encephalographic studies were made in 12 cases. A craniotomy was done in 15 cases and autopsies were done in five. Ten were tested with the Westinghouse 2A audiometer and shown to have no hearing defect which would account for the symptoms. The gnostic ability, that is, the ability to recognize common sounds, such as the ringing of a telephone, was not routinely tested because of inadequate test material. However, general observation of their behavior about the ward showed that most of them reacted with understanding to sounds other than speech. Recently, a technique for testing this gnostic ability has been worked out by Dr. Dozier and, at present, is being used routinely for the patients showing word-deafness.

Of the 17 patients, 14 were purely right-handed individuals and had tumors of the left temporal lobe. One was a left-handed individual with a tumor in the right temporal lobe. Two were people who showed mixed motor patterns and had right temporal lobe tumors.

All of these patients had detailed and elaborate examinations of the language function. However, for the purposes of this discussion, only the results of the common object series will be considered. This test was selected because it involves the use of single words, so common that it is fair to assume that any patient has, at one time, been able to use them freely. The same test words are used to test each aspect of the language mechanism; that is, naming or spontaneous speech, repetition, understanding of spoken word, reading aloud, understanding of printed word, copy, writing from dictation and spelling. This permits a comparison, first of one aspect of language function with another aspect in the same individual; and second, of the relative disabilities of different patients.

The technique—a modification of the head serial aphasic test No. 1—has recently been described in the *Bulletin of the Neurological Institute of New York.*[4] In order to show how

some of the graphs were obtained, I shall review it briefly. Six common objects—a key, a pencil, penny, matchbox, scissors and a comb—are presented in the order named. The patient is first asked, "What is this?"—that is, to name the object. Each object is presented three times. This is done for each of the language functions enumerated above. The understanding of the spoken word is tested by displaying the six objects and asking the patient to "show me the key," "the pencil," "the penny," etc., until each object has been asked for three times. The patient, then, has 18 chances at making a selection. Any hesitancy or confusion or wrong selection is accounted as an error. If the patient is credited with nine errors, it is calculated that his understanding of the single spoken word is reduced by 50 per cent. In other words, he has a 50 per cent word-deafness. This same sort of percentage reduction is calculated for each aspect of language (see Table I).

TABLE I.

Case No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	
Correct	10	8	8	16	17	9	12	3	7	0	10	5	10	10	6	7	0	138
Pause—Correct	4	0	3	0	0	5	0	0	0	0	7	0	0	2	0	4	0	25
No Pause—Wrong	0	5	1	2	0	1	6	15	5	18	1	5	4	0	4	3	12	82
Pause—Wrong	4	5	6	0	1	3	0	0	6	0	0	8	4	6	8	4	6	61

Showing type of responses made to the single spoken word.

Table I has been prepared to show the type of error which was found in these patients under consideration. It will be noted that some of the patients are credited with errors when they showed hesitancy, even though the correct object was eventually selected. The reason for this is readily seen when it is remembered that such objects as a key, a pencil, penny, etc., are so well known to almost any patient that even a slight hesitation must be considered significant.

As the figures in the table show, only two of the group showed a total inability to understand the spoken word. Some understood far better than others. However, it must be remembered that for this test it is only necessary to under-

stand a single word. All of these patients showed great difficulty when slightly more complex material was used—for example, none of them was able to execute a two-commission command, such as "you take the scissors and give me the pencil." On the other hand, even those who showed great difficulty in these tasks appeared, upon occasions, to understand quite well remarks of others. This was particularly true when such remarks referred to the patient (see Table II).

TABLE II.

% Loss	COMMON OBJECTS						
	Naming	Repetition	Oral Reading	Spoken Commands	Printed Commands	Writing Dictation	Spelling
0	——	xxxxxxxx	xxx	——	xxx	——	x
		x					
10	——	x	——	——	——	——	——
				x			
20	——	——	xx	——	——	xx	x
30	x	xx	xx	xx	x	——	——
				x			
40	x	x	——	——	——	——	——
				x			
50	——	x	xx	x	xx	x	xx
		x					
60	——	——	——	xxxxx	x	——	——
	x				x		
70	——	——	——	——	x	x	——
	xxx		x	x	x		x
80	x	——	——	xx	x	——	——
	x		x	x			
90	xx	——	——	——	——	——	——
100	xxxxxxx	xx	xxx	xx	xxx	xxxxxxxx	xxxxxxxx
						xx	x

Graph showing the relationship of word deafness to other aspects of language—common object series.

Inasmuch as the entire group showed disability in other aspects of language, Table II has been prepared to show how the reduction in understanding the spoken word compared with the reduction in other aspects. It will be noted that the average of the group had great difficulty in naming the object shown, were able to repeat the name fairly well and showed moderate confusion in understanding the spoken word. Both

the ability to read the printed name aloud and to select the object from the printed name were impaired, often to a degree as great as the ability to understand the spoken word. The majority had their greatest difficulty in spelling and in writing the name of the object. This table is used to show word-deafness in its proper setting; that is, as a part of an aphasic state; or, more narrowly, as a part of a sensory aphasia.

It may be of interest to examine more closely the spontaneous speech of these patients. Those who were most severely affected were unable to produce speech which was understandable to others. Such speech has been characterized as jargon speech. Many who were less severely affected gave utterances which, in part, could be understood by others. A few examples may serve to illustrate the character of this speech:

1. During the testing of visual fields, one patient said, "I see your lamping but can't see your white;" by which he meant, "I see your hand but can't see the white object."

2. Another said, "Don't let them tell you about my cigarette." He meant, "Don't you tell about my cigarette."

3. And another, "It drops out of my head all of a sudden," meaning "I suddenly forget it."

In the above examples the interpretations are those of the observer. It was felt that the circumstances in which the comments were made permitted these interpretations. This element of personal interpretation is eliminated when the patient is subjected to routine testing and asked to name the common objects. Here, the response is to a single object and there is little doubt as to what the patient is trying to say. Responses elicited in this way are tabulated in Table III.

This table shows one important characteristic of the disability of these patients; that is, the inconsistency of their responses. One patient, for example, on three attempts to name a penny called it "a wala" the first time, "a penny" the second time, and "a sinkel" the third time. When these same terms, which the patient himself produced, were said back to him, he showed that they meant no more to him than any other words; that is, when he was asked to select "a wala" (his own word for penny on the first attempt), he picked up the key.

TABLE III.

Responses made in naming the objects seen—"jargon speech."*

KEY

lamp
tē mi
kā nʋ pen yʋ
lʋm brelʋ
bʋ śhʋl
tū ā stlʋā
kī beiśh
dōr
yō riŋ
klī wiŋ
pes kī

PENCIL

wār tin
kər nʋm
lam pi
hō slō
am blaba
ā tū bai fōr
ā smāl mārk pensil
nʋfa mī yā
nai fər
bed bāl
pen slē
spin sil

PENNY

wālā
wun sinkl
simu
sū vā
pa ni
tak tak
mis tə yer
kept

MATCH-BOX

Mas ā tūr
van ti gār
ma ćhəʒ
pinə
neks pil
val kām
kōn stan
sʋm ṫhiŋ tū ripōrt en,
sigarets er sʋmṫhiŋ
baćh lō
beks neks
man ćhiŋ

SCIṠSORS

adami
ma ćhəʒ
ćha kʋ
sma sʋ
tū bl beks
pasiv
tū sent stam
spi nis
siʒ wa
śh arʒ
a śharpnar

COMB

grā fʋ
maćh fer mai her
si wa mʋ
śh ipʒ hed
pinsa yū rait it en

*The system of diacritical markings here is according to the revised scientific alphabet as used in the New Standard Dictionary.

The attitude of some of these patients toward their auditory confusion is revealed by their comments made during the test period. One patient, when asked to select these objects from the spoken word, said, "They don't mean a damn thing

to me;" and another in the same situation said, "I can't tell one from the other today;" a third, when asked, "What is your name?" said, "Name what? You say name—name what?" When asked his age he said, "What is my age? Is that what you said?" When answered, "Yes," the patient replied, "What is age. I don't know what age is either." Another, whose native tongue was German, but who, prior to his illness spoke several languages fluently, was observed talking with his family. They were speaking in German. His replies were a jumble of English and German. After several minutes of conversation he remarked, "I don't quite get it," and turned to ask the examiner whether his relatives were speaking English or German. It must be remembered that these same patients who had difficulty in selecting these objects when they heard the name were able to demonstrate that they knew how to use the object.

Word-deafness, then, should be considered as a disability in the language function which occurs only in conjunction with other disabilities in language. Of the aphasic cases studied, word-deafness has rarely been the outstanding language defect, even though the pathology was limited to the temporal lobe. This does not mean that the finding of word-deafness has no diagnostic or localizing value, for while word-deafness does not appear uncomplicated by other language disabilities, there are many cases of motor aphasia with little or no word-deafness and cases of sensory aphasia where the word-deafness is negligible as compared to the word-blindness. In such cases it is found that the lesion does not involve the temporal lobe. In former cases, the lesion is to be found anterior to the temporal lobe; in the latter cases, posterior to it. The finding, therefore, assumes a diagnostic value when it is considered in relation to the other aspects of language and its presence is indicative of damage to the temporal lobe of the dominant hemisphere.

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FROM AFFECTIONS OF THE BRAIN.

(b)—PSYCHOGENIC FACTORS (HYSTERICAL AND EMOTIONAL).*

DR. MILES ATKINSON, New York.

I think perhaps we as otologists do not take sufficient cognizance of the psychogenic aspect of hearing, one of the most important sides of which is adaptation. If there is any interference with the hearing mechanism, whether by disease or by congenital malformation, some degree of deafness is produced and it depends very much on the individual how much he or she will compensate for that. The young child will compensate very readily, as he will compensate very readily for the loss of any function. If he loses an ear or part of an ear (whereby I mean the hearing, of course) he compensates, and it must have been everyone's experience at one time or another to come across a child who has had a radical mastoid operation and yet has surprisingly, very surprisingly, good hearing in that ear. If we go to the other end of the scale, there is the old man who, by reason of his age, possibly through senile neuritis or whatever it may be, is beginning to go deaf, though by audiometric and tuning fork tests the diminution in the amount of his hearing is not apparently very great. He says that he cannot hear, that he has the greatest difficulty in hearing conversation, and his trouble is that, apart from being an old man (and you can't teach old dogs new tricks), he hears only sound. He can hear simple sounds, but he is unable to make those sounds into a pattern, the pattern of words and sentences.

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For simplicity, I like to think about the auditory tract in the psychogenic respect in a simple way, dividing it into two parts. The first portion goes from the external organ of hearing to the centres in the midbrain and is responsible for what one may call basic hearing. It is along this, I imagine, that ordinary simple sounds, the sounds of tuning forks and the audiometer, are heard. And then there is a higher, cortical centre, connected with the basic midbrain centre, a cortical control for intellectual hearing, which sorts out those sounds, fixes them together into a pattern, makes words of them, and thus we have words, memory, and so forth.

The first, the basic hearing, is what we test with tuning forks; the second is intellectual, and on the ability to hear intellectually depends the degree of deafness in many patients. In this way you have the young person, who is easily adaptable, and it doesn't matter if he hears wrong, for he soon adapts; and you have the old man who cannot adapt himself. He hears all his s's, the high tones as f's, and he can't get accustomed to hearing the word "ship" as "fip." It doesn't sound right to him. He never has heard it like that, and he is too old to learn to hear it like that.

In between are people, some of whom have a high degree of intellectual hearing, who sort out their new sounds readily and learn to understand this different code of syllables they hear, which is in reality another language. These are the people who learn a foreign language quickly—they have an ear for languages. Then there are others in the middle class who, with apparently a very small (and we have all of us seen them) lesion of the ear, have a very great interference with hearing. Those are the people, I suppose, who, as far as their hearing is concerned, are intellectually slow. They have not a faculty for hearing or for listening. It is something in their make-up; they can't help it. There are some people who can't learn anything. They have tied their tie in the same way all their lives and they are going to go on tying it in the same way. They cannot learn and so they are unadaptable, and people of that sort become deaf with a very small lesion in the hearing apparatus.

Then there is another psychogenic factor. There is a large group of cases that we call functional or hysterical—we saw many of them during the War. They were all grouped com-

fortably under the nice omnibus term, "shell shock," and some of these shell-shocked people lost their hearing. In civil life the hysteric is usually a person of unstable mentality, an unstable mental system based on an hereditary defect. They have an ill-bred mental system, possibly a malco-ordinated and maleducated mind, which has never been taught to discipline its life or thoughts, and those people will be particularly prone to suggestibility and to all the functional disorders that arrive. They may become deaf. There is so often a feeling in our minds, I think, that patients suffering from functional disorders, disorders which we cannot place on any organic basis, are rather despicable. But they are in fact no more despicable because they have a poor nervous system than the patient who has what used to be called a tubercular diathesis, who is particularly susceptible to tubercle. They are rather to be pitied than despised, for, after all, we are all potential hysterics.

If you produce an insult to the nervous system sufficiently forcibly and sufficiently frequently, over a sufficiently long period of time, the person whom you insult in that way will crack—and so it happened time and again in the War. As Kipling put it in the poem many of you may have read, "My Mother's Son":

> "What with noise, and fear of death,
> Waking, and wounds, and cold,
> They filled the cup for my Mother's son,
> Fuller than it could hold."

If it goes too far, our cups can be filled fuller than they will hold, so that we are all potential hysterics. We should, therefore, have a fellow-feeling for such patients, and realize that the hysteric is a truly ill person. He is quite convinced of his own trouble, not like the malingerer, who is often associated with him in the medical mind. The malingerer is quite conscious of what he is doing, while the hysteric is not acting purposely, he does not know what he is doing. The one cannot be frightened by a threat, the hysteric; the other, the malingerer, can be and is. The malingerer can be caught by a trick and often is; the hysteric cannot. But the hysteric can be cured, often very dramatically, by a sufficient force of personality, and in particular by reproducing the localized suggestion which has made him produce his

functional disorder. For instance, if a sudden shattering sound has produced a deafness, that deafness can be cured by a similar stimulus, by another sound.

I should like to tell you of a rather interesting instance of that. I was dining with a friend of mine in London during the War, in a restaurant, and he had been "blown up." He was a very good shell shock case, and he was very deaf. He had been "blown up" by the explosion of a "Minnie," and you will remember the noise they made, and at this time when I was having dinner with him, it was three months afterwards and he was still extremely deaf. There was a silly trick at that time which some of the bright young things had of taking a match out of a matchbox, a square match, and flicking it through their fingers so (demonstrating), beside the ear, and when that happened (and I had it done to me), it sounded for all the world like a bullet going "ping" past. Well, we were sitting having dinner and one of the bright young things in London at that time did this to him, and he was up and out of the chair and out of the door, muttering imprecations, and you couldn't see him for dust, but, when we did get him back, he could hear. He had been cured by the same stimulus.

There were many cases during the War, and after the War, of deafmutes who saw something on a movie and were so emotionalized that they stood up and talked. It was an almost every-day occurrence for such a stimulus to produce such a result.

The malingerer isn't like that, he knows perfectly well he isn't deaf. So perhaps I may finish with one story about a malingerer, a rather interesting case. During the War there were fashions in disease, simulated disease, to avoid going to sea or to the trenches, and we used to sit on boards on these men. At this time the fashion was deafness. There was one man who had been assigned to the Grand Fleet and didn't at all want to go. We were quite sure he was faking what he called his "gun deafness," but it wasn't too easy to be absolutely certain about it. So we put him in the hospital and I used to go around with the orderly every day and order for him all sorts of delicacies on his chart. One day he would have cream with his stewed fruit, another day I would say, "You had better have some chicken today,"

and so forth, the orderly having been told all the time to give him nothing but milk. We just talked in an ordinary voice and this man was very deaf, so he couldn't possibly have heard, and every day the orderly would say to me, "Yes, he had that, sir, and enjoyed it very much. Now, what do you think of giving him so-and-so today?"

Well, he stood this for about a week and then one day I went around and the orderly said, "Oh, he had a fine dinner yesterday. He did enjoy it."

I said, "That is very good."

And he couldn't bear it any longer. He said, "It isn't true, sir! It's a bloody lie! I've had nothing but milk for a week!"

AMERICAN LARYNGOLOGICAL ASSOCIATION CASSELBERRY PRIZE FUND.

The sum of $500 having accrued from the Casselberry Fund for encouraging advancement in the art and science of laryngology and rhinology, said sum is now available, in part or as a whole, for a prize award, decoration, or the expense for original investigation or research in the domains mentioned above. Theses or reports of work must be in the hands of the Secretary, Dr. James A. Babbitt, 1912 Spruce street, Philadelphia, before Feb. 1 of any given year.

SYMPOSIUM.

THE NEURAL MECHANISM OF HEARING.

V.—ETIOLOGICAL AND CLINICAL TYPES OF SO-CALLED "NERVE DEAFNESS."

MALOCCLUSION AND ITS RELATION TO EAR AND TEMPOROMANDIBULAR DISORDERS.*

DR. WM. H. CRAWFORD, New York.

Dental cripples are very common. The following information is taken from a report by Dr. P. J. Brekhus[1] on a statistical study of the loss of human teeth. According to his figures computed on a survey conducted on 14,935 patients in 1933 to 1935, 35.5 per cent of the upper first molars were missing in the age group of 31 to 40 years. Forty-four per cent of the lower first molars were missing in the same age group. In the age group between 41 and 50 years, 45.5 per cent of the upper first molars were missing and 52 per cent of the same teeth below were missing. This is to mention but one tooth.

This alarming situation is mentioned because it has a definite bearing on the subject I have been asked to discuss. The first permanent molars are the key teeth to the dental arch, and when they are missing the teeth posterior may drift forward and those anterior may drift posteriorly. This holds true also for any missing tooth. The loss of teeth and the subsequent drifting of those remaining reduces the distance between the maxilla and mandible, and produces the so-called "closed bite." This condition is, therefore, very common, as the loss of teeth is the most common cause.

If vertigo, loss of hearing and tinnitus are commonly caused by overclosure, we are living in a dizzy age, and this we all know is not so in the true sense of the word. According to statistics, the loss of teeth is decreasing and, therefore,

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these disorders should, if they are caused by closed bites, also be decreasing. This is doubtful. If only a very small percentage of patients who exhibit a closed bite condition are suffering from the conditions mentioned above, then it seems to me quite natural to feel that the correction of overclosure will not always offer a solution to temporomandibular disorders.

However, nothing is lost by attempting to correct these conditions by opening the bite, providing it is done in such a way as not to injure the supporting tissues of the remain-

Fig. 1. (a) Holliday modification of Bullett mastoid apparatus, showing patient in position.

ing teeth. Our experience teaches us that the bite cannot be raised on one side alone, or on only a few teeth, but that the mandible functions as a unit and if one side is opened, then both sides must be opened. The older the patient, the more important this rule becomes. Natural solid teeth are important in this work and each tooth used to open the bite must be treated to function in harmony with the other remaining teeth, or it will soon be lost through trauma.

As early as 1920 cases were repored by Wright[2] where patients' hearing was improved by bite-opening. Costen,[3]

Goodfriend[4] and others have reported more recently many more cases where patients were benefited by bite-opening procedures.

Patients suffering from malocclusion and overclosure who have come under my observation may be divided roughly into four groups. The first, and by far the largest group, are those patients who are suffering from no other apparent illness except loss of masticatory function and poor appearance. The second, and next most common, are those patients presenting malocclusion with overclosure and complaint of pain in or near the mandibular joint, pain posterior to the

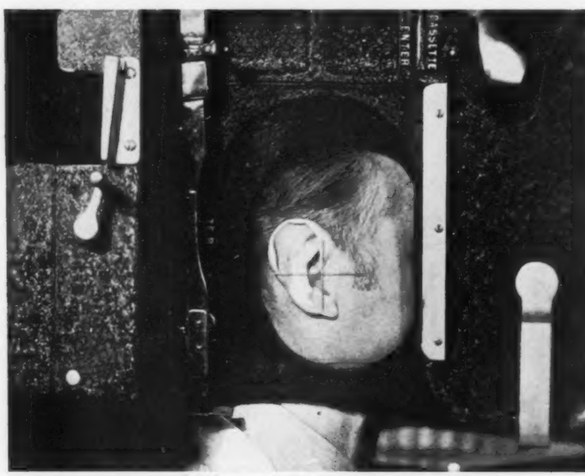

Fig. 1. (b) Holliday modification of Bullett mastoid apparatus showing method of positioning head.

ear, clicking and rasping of the condyle in opening, headaches and pain, especially after eating or early in the morning. Pain in these cases may be due to an upward and posterior displacement of the condyle bringing pressure on the chord tympani and auriculotemporal nerves. It is my opinion that pain in this region may also be due to injury to the ligaments supporting the mandible. Some patients definitely associate the beginning of their difficulties with trauma suffered at the time of the extraction of a tooth by forced opening of the mandible. Patients in this group are fre-

quently aided by bite-opening. We have treated many of these cases successfully. These patients also sometimes complain of a temporary loss of hearing that may be corrected by swallowing or by depressing the mandible. The third group are those complaining of a lack of hearing without pain. To date I have not treated any of these cases successfully. The fourth group are those suffering from vertigo. Several of these patients have been treated successfully by bite-opening. Others have been benefited temporarily only.

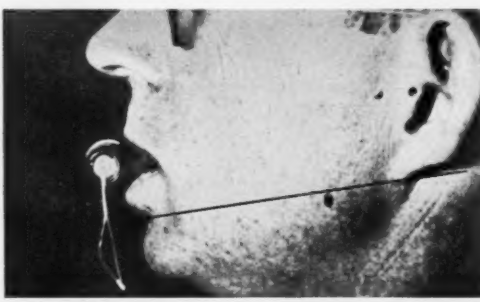

Fig. 2. Light tracing on photographic emulsion showing opening movement of patient with normal centre of rotation below or approximately level with the attachment of the sphenomandibular and stylomandibular ligaments.

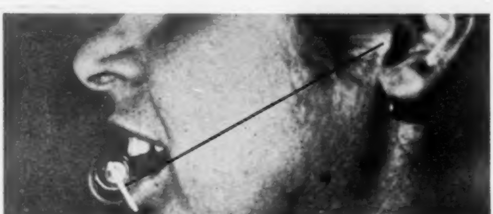

Fig. 3. Light tracing on photographic emulsion showing opening movement of patient with abnormal centre of rotation about head of condyle.

The X-ray has been used extensively in an effort to find a way to determine if the condyle was in an abnormal position in the glenoid cavity. The apparatus used for this purpose is the Holliday modification of the Bullitt mastoïd apparatus shown in Fig. 1, in which the patient, plate and tube are always in a fixed position. The patient is in a vertical position. Plates of subjects with a normal complement of teeth

indicate that the condyle may be in a large variety of positions in the glenoid cavity. The space occupied by the articular disc is not constant. This is true also in all the cases where there was malocclusion and overclosure. The picture in these cases is not dissimilar to the normal occlusion groups.

I have found that in cases of normal occlusion any opening of the mandible causes the condyle to change its position in the glenoid cavity downward and forward. This brings the rotation point or area of the mandible to a point on a level with or somewhat below the attachment of the stylomandibular and sphenomandibular ligaments (see Fig. 2).

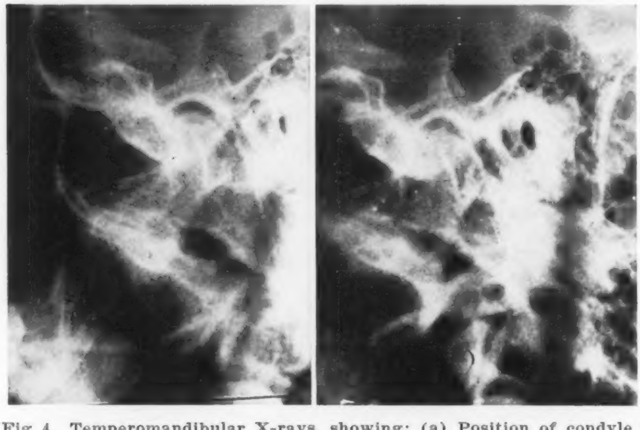

Fig. 4. Temperomandibular X-rays, showing: (a) Position of condyle in glenoid cavity on right side with teeth closed; (b) position of condyle in glenoid cavity on right side with mandible depressed as far as possible.

This is not necessarily true in cases of overclosure. In many of these cases the mandible may be opened considerably without drawing the condyle away from the posterior boundary of the glenoid cavity. The rotation in this case is actually in the head of the condyle and the stylomandibular and sphenomandibular ligaments do not function to draw the condyles forward. This condition is illustrated in Fig. 3 and also in Fig. 4, showing the position of the condyle with the mandible closed and open to its full extent. This has a definite bearing on positioning the condyle to relieve possible excessive pressure on nerves located in this area. In order to

relieve posterior pressure in these cases the mandible must be moved forward, as well as opened, and held in that position by interdigitating cusps.

CONCLUSIONS.

1. Most cases exhibiting considerable overclosure and malocclusion do not complain of pain, loss of hearing or vertigo.

2. Pain in its various manifestations is frequently relieved by opening the bite.

3. We have not effected to date any permanent improvement in hearing.

4. Vertigo has in some cases been effectively relieved.

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SYMPOSIUM.

THE NEURAL MECHANISM OF HEARING.

V.—ETIOLOGICAL AND CLINICAL TYPES OF SO-CALLED "NERVE DEAFNESS."

(b)—"NERVE DEAFNESS" OF KNOWN PATHOLOGY OR ETIOLOGY.

SIMULATED DEAFNESS (MALINGERING).*

DR. DOUGLAS MACFARLAN, Philadelphia.

Dr. Fowler has wisely chosen the title, "Simulated Deafness," which we have commonly called malingering, in assigning me a subject. The choice of the adjective "simulated" is a good one. We must remember if it was only for the sake of detecting malingering, the usefulness of our malingering tests would be very limited; there are few of these cases that we are called upon to expose. On the other hand, many of our cases of deafness are unwillingly inaccurate in their reporting under our testings. It is obvious that we have largely to depend upon this patient-reporting to get recordable results, for hearing-examination is more subjective than objective. We, then, must find the true service in a knowledge of this subject in its help in checking the accuracy of patient reporting.

I would venture the comment that one can judge the ability of an otologist as to his hearing-testing by his familiarity with methods of detecting errors in patient-reporting. And this familiarity of the subject does not depend upon a knowledge of the many malingering tests described in the literature. Let us take, to illustrate this point, some commonplace observations.

Case: You are taking the fork hearing time (A.C. and B.C.) on a patient and the repetitions of the tests show a wide variation in the time reported. What is wrong? An

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intimate knowledge of the subject tells you that one of a number of factors may be at fault: *1*. Varying noise level may be changing the dampening of the sound. *2*. Varying technique by the tester may be changing the intensity of vibration. *3*. The patient may be exhibiting tone memory, so that he cannot accurately report the end-point of his hearing. *4*. With the testing of children, one should add the attention factor.

But you want an accurate report for recording. And so you set about to cover each of the above items. *1*. You make sure that your noise level remains relatively constant by selecting a quiet testing place; you exclude most of the sound by stopping the untested ear; if there is hearing around the head you dampen this ear. *2*. You are careful to maintain an identical technique in all your retestings. *3*. You use the "on and off" application of sound, so as to avoid tone memory. You may even go so far as to ask the patient to hum the tone that they hear, so that you may be sure that they are reporting upon the testing tone and not on its octave overtone nor on a tinnitus that may be present. *4*. You sharpen up the patient to be attentive to hearing by emphatic instructions before testing.

Such methods may indeed be miscalled malingering testings, but they are surely the means of getting accurate reportings as to actual, not assumed deafness.

The accuracy of the reportings of repeated fork-time hearing tests are an index of the dependability of the patient's reports.

A convenient but relatively crude check is the stopping of the E.A.M. after the patient has reported the end-point of B.C. hearing. Hearing should return.

Many, if not all, methods of dampening can be used to invent a great variety of malingering tests. If the patients hear above the dampening, their voices are raised, especially if there is some degree of catarrhal deafness. Exclusion of the hearing of the untested ear at critical times allows you to determine whether or not the tested ear hears. This test can be performed by the selection of sentences with key words which must be heard to have the sentence make meaning. The key words are dampened out of the untested ear while the sentence is being spoken.

Keiper's Voice Test: The doctor approaches the supposedly deaf patient from a distance of 20 or more feet after having asked the patient to raise the hand as soon as he hears the voice.

Barany's noise apparatus in an ear makes a hearing person raise the voice. A piece of paper rattled at the ear of a person reading will do the same.

Dampening is nothing more than sound masking, equivalent to sound exclusion. Many tests are of the exclusion type.

The classical dampening test is, of course, Stenger's test, with which you are all familiar. Two forks of like pitch are used, and an identical but louder tone is thrown into one ear to "kill" the hearing of tone in the other ear. If the patient reports the disappearance of tone to which he has been listening, he must have had hearing in the ear into which the dampening pitch was thrown.

Among the tuning fork tests is that one in which malingering is detected by the patient's accuracy in timing. A patient cannot wilfully select a shortened timing. You should run through repetitions of the timing, varying intensities of the fork, checking it with your own hearing and throwing in interruptions that make it still more difficult to be consistently inaccurate.

I have already suggested that modification of the Weber test, where the meatus of the tested ear is stopped by the finger, after the B.C. end point is reached, B.C. hearing should return.

The use of one vibrating fork and one nonvibrating fork, confusing the patient as to his A.C. and B.C., is often effectual, though without properly thinking the problem out, one often finds the examiner as confused as the patient.

Confusion tests with the bell stethoscope and tubes leading to the patient's ears are very simply devised by pinching the tubes.

Plugged Stethoscope Test: The tips of a stethoscope can be removed and one plugged with wax. They can be alternately used in the good and "bad" ears to trap the patient.

Galton Whistle Test: If a low calibration of the Galton whistle be taken, the patient should hear the sound "around

the head" and in the good ear when the whistle is put to the "deaf" ear. He will usually report no hearing.

In dealing with deafness beware lest lip-reading enters in to deceive you.

Dr. Fowler's paper on intensity matching by the two ears suggests an excellent test, for here, surely, it would be difficult to simulate. Remember, for your own accuracy, in testing the single ear by bone conduction that the skull impedance is often only 5 to 10 dcb., and the patient may be truly reporting hearing from the deaf side, but by reference, which neither he nor you may recognize.

Do not overlook the value of the unilateral vestibular tests as confirmatory evidence of considerable value, and in unilateral nerve deafness, which has involved the entire end-organ or VIIIth nerve (such as in basal fractures), do not overlook the possibility of unilateral vestibular accommodations.

As to hysterical deafness, it seems reasonable to consider it comparable in every sense except cause and cure with actual deafness. There is a subconscious negation of all stimuli to hearing. Even a very loud sound does not evoke the pain sense. This latter point I believe to be an important observation in the attention-deaf child.

I cannot leave the subject of testing simulated deafness without mentioning the testing of the little deaf child, deaf before language. Has he or has he not some residual hearing? Is this a usable amount? Is he perhaps an attention-deaf case? Is he reporting tactile sense for hearing? I have elsewhere described a fork method of discriminating between tactile sense reporting and hearing. It depends upon the use of the interference zone of the fork. Better still is to have the parents train the child in the conditioned response of raising the hand when there is presented a sound loud enough to reach the tactile sense. When the child is trained, the otologist may drop the intensity away from the tactile threshold, when, if the child responds correctly he must be reporting hearing.

The remarks have all been somewhat sketchy, due to the brevity of the paper; I trust, however, that they show the real uses of an understanding of simulation for our routine testing needs.

1805 Chestnut Street.

SYMPOSIUM.

THE NEURAL MECHANISM OF HEARING.

V.—ETIOLOGICAL AND CLINICAL TYPES OF SO-CALLED "NERVE DEAFNESS."

(b)—"NERVE DEAFNESS" OF KNOWN PATHOLOGY OR ETIOLOGY.

FAMILIAL AND DEVELOPMENTAL DEFECTS OF NERVE DEAFNESS.*

DR. MAX A. GOLDSTEIN, St. Louis.

The approach to this subject is a difficult one, as the available data is meagre and not uniformly and systematically classified, and, where living subjects are reported, the laboratory diagnosis and corroboration is usually absent.

In 1921, I presented to this Society a subclassification of congenital deafness as observed in the familial history of large groups of young deaf children. My subclassification consisted of: *a.* Biological Congenital Deafness; *b.* Pathological Deafness.

Biological Congenital Deafness: In Group A, I classified all biological and anatomical irregularities in growth as they may affect the temporal bone, with special reference to such anomalies in the labyrinth. These included: Absence or developmental malformation of the helicotrema of the cochlea, malformations of the cochlear whorls and an especially cited case by Adam Politzer of the branches of the cochlear nerve as supplied to the neurohistological structures penetrating the organ of Corti. In this case, the nerve sheath was present throughout all the cochlear nerve branches, but no neuroglia was in evidence.

Then, too, there must be included in this classification underdevelopment or retarded growth of the auditory field in the cortex. While such an anatomical defect is theoretically admissible, practical evidences in the actual microscopic exam-

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ination of the brain tissue in the auditory field have not yet been satisfactorily demonstrated; however, there is enough clinical and macroscopic observation to warrant inclusion of such data in the discussion of the etiology of biological congenital deafness. Inference to be drawn from such data would suggest: *1.* Anatomical deficiencies or anomalies in the peripheral or end-organ of hearing in the labyrinth, with a perfectly normal auditory field in the brain cortex; *2.* Normal peripheral end-organ of the cochlea, with a defective development of the auditory cortex; *3.* Defective end-organ, plus defective auditory cortex.

Pathological Congenital Deafness: Here we must consider all changes of pathological character that may take place during fetal development or at the time of birth and should include hereditary syphilis, rickets, quinine and other drug toxemias, intrauterine hemorrhage and birth injury.

I have seen a number of cases of profound nerve deafness in young children where the familial history is definitely luetic, where some residuum of hearing is present and where, in the course of a few years, such residual hearing is gradually snuffed out. In several instances modified Salvarsan therapy has eliminated the luetic inroads and the residual hearing has been retained.

It is interesting to report at this time that in two such cases the blood Wassermann was reported negative, but on spinal puncture the laboratory report showed a positive Wassermann. These cases would have remained without luetic therapy if this unusual diagnostic measure had not been carried out to its extreme limitation.

The interesting reports of H. Marshall Taylor on "Quinine Deafness in Young Children of Malarial Mothers" offer another angle to the pathological etiology of nerve deafness.

Last year, in my report on "Genetics in Otosclerosis," I raised the question whether many of the cases which come under our observation may be considered as early otosclerosis rather than as congenital deafness. This differential diagnosis is a difficult one and, in the light of the findings of Gustav Alexander and Stacy Guild, it must be recognized that otosclerosis can begin at a very early age and the possibility of otosclerosis must not be lost sight of. If oto-

sclerosis can be developed in embryo we have another confusing problem for solution, in that many of the cases diagnosed as congenital deafness of unknown origin may be found to be otosclerosis in the embryo. Our findings are still too meagre in this field to discuss this question more definitely.

The difficulties in making a diagnosis of deafness in the young child are still too numerous to afford us a definite form of approach in this somewhat unknown field. We must depend on further research, larger numbers of clinical and laboratory corroborations, a more careful study of the brain cortex and a more positive form of functional test.

I have confined these brief observations entirely to the young child.

4574 West Papin Street.

SYMPOSIUM.

THE NEURAL MECHANISM OF HEARING.

V.—ETIOLOGICAL AND CLINICAL TYPES OF SO-CALLED "NERVE DEAFNESS."

(b)—"NERVE DEAFNESS" OF KNOWN PATHOLOGY OR ETIOLOGY.

THE EFFECT OF ROENTGEN RAYS UPON HEARING. A PRELIMINARY REPORT.*†‡

DR. V. Y. KASABACH, New York.

LITERATURE.

The effect of Roentgen rays and radium on the hearing and tinnitus has been studied since 1905. Ewald[1] (1905) and Marx[2] (1909) used radium implantation near the labyrinth of animals and recorded the effect upon that organ. McColloch[3] (1913) reported four patients with extreme deafness who received Roentgentherapy. He concluded that there was a slight but definite improvement of the hearing. Sobotky[4] (1917) reported 50 cases with deafness and tinnitus who showed improvement following irradiation. Jarvis[5] (1923) tried small doses of Roentgen rays on a series of cases. He found it effective in alleviating tinnitus and valuable in treating aural disease. McCoy[6] (1923) reported a series of 45 cases. Of these, 36 showed definite improvement in hearing. The symptom of tinnitus was relieved in 16 cases. More recently, Desjardines[7] (1931) reviewed the literature on this subject. He emphasized the lack of experimental data, and on the basis of clinical observation he believed radiotherapy rendered useful and, occasionally, "great therapeutic service."

Girden[8] (1935) conducted a series of experiments and studied the effect of Roentgen rays on the hearing of 12 dogs.

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†From the Department of Otolaryngology of the Presbyterian Hospital and of the College of Physicians and Surgeons, Columbia University, New York.

‡This study was made possible through the co-operation of the members of the Radiologic Staff. I am indebted particularly to Dr. G. R. Brighton and Dr. Haig Kasabach.

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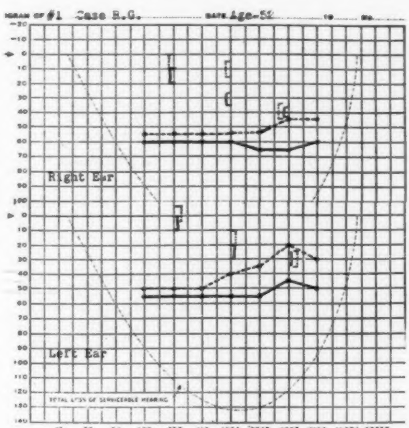
Patient	Age	Diagnosis	Radiotherapy — 200 KV Machine — Used Dosage	Audiograms and Period of Followup	Present Status of Patient	Results of Radiotherapy
1—R. G.	52	Cylindroma of nasopharynx	1,200-1,350r over each of 6 different fields centering on nasopharynx	Jan., 1937, to May, 1937	Definite reduction in the size of the tumor. Patient in a weakened state	There is definite evidence of improvement of the air and bone conduction in comparing the audiograms
2—J. C.	34	Lymphosarcoma of nasopharynx	3,000-4,000r total dosage given over several fields	Oct., 1936, to April, 1937	Mass in nasopharynx smaller. The metastatic mass in neck not improved	There is appreciable change in the air conduction, but no change in the bone conduction
3—E. W.	39	Squamous cell epithelioma of nasopharynx	Total 3,200r from 8/6/36 to 9/5/36. Since has had therapy to metastatic lesions	Aug., 1936, to May, 1937	Nasopharyngeal mass gone. Metastases to spine, leg, rib, and right ileac region	Definite improvement in hearing, especially the bone conduction
4—J. D.	18	Capillary hemangioma involving entire nares	3,500r total over several fields; 1,800r through cone intranasally	July, 1936, to May, 1937	Mass definitely shrunken in size. Destruction of septum and small perforation in palate	Slight improvement in the air conduction. Definite improvement in the bone conduction
5—A. R.	34	Basal cell epithelioma of preauricular skin	5,000r total over left preauricular region. Right side not radiated	March, 1937, to May, 1937	No recurrence of the lesion seen	Improvement of the bone conduction, especially on the left side
6—F. P.	17	Squamous cell epithelioma of nasopharynx	2,000r over each of 5 fields. Total irradiation, 10,000r	March, 1937, to May, 1937	Reduction in size of tumor. Dermatitis of skin and ext. auditory canals	Slight improvement in both air and bone conduction. The left slightly more so.
7—E. E.	61	Squamous cell epithelioma of right antrum	4,500r over several fields	Sept., 1936, to Jan., 1937	Patient in the terminal stages of the disease	Definite reduction in the acuity of hearing as the patient's health grew worse
8—W. B.	46	Squamous cell epithelioma of right tonsil	3,000r given over the right side only	Aug., 1936, to Dec., 1936	Patient showed no improvement. Died 1/22/37	Definite reduction in the acuity of hearing
9—J. B.	53	Squamous cell epithelioma of nasopharynx	3,000r from 10/18/35 to 12/4/35; 2,400r from 12/12/35 to 1/27/37	Oct., 1935, to March, 1937	Tumor disappeared. Involvement of the base of skull. Died 3/5/37	Definite reduction in the acuity of hearing

In summarizing his results he stated, "there resulted a transient gain in acuity of about 5.5 dcb., lasting from two to five weeks. In no cases was hearing impaired."

MATERIAL.

In analyzing the effect of Roentgentherapy on the hearing by means of the audiometer, it is emphasized that this report is only preliminary. For the purpose of this study the material is classified into three groups:

Group I: This consists of a number of patients who were treated in the radiotherapy department. The majority of the cases had a neoplasm in the nasopharynx or near the region of the ear. The field of radiation was so arranged



——Solid lines indicate-Before treatment
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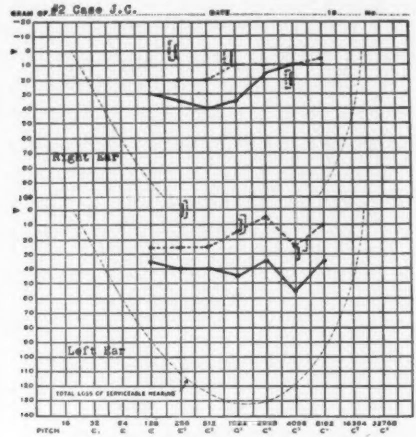
as to treat the primary lesion. In order to accomplish this, one or both ears had to be included in the irradiated area.

Group II: A selected group of cases who have come to the Ear, Nose and Throat Clinic because of defective hearing. The examination revealed air, bone or mixed type of deafness without any evidence of active infection.

Group III: This group includes a series of patients with clinical manifestations of chronic otitis media, otitis externa, granulations and eczematous lesions in the external auditory canal.

In this preliminary report, only the cases falling in Group I will be presented. The results of the remaining two groups



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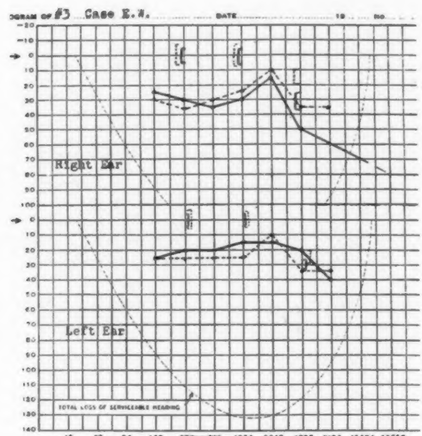

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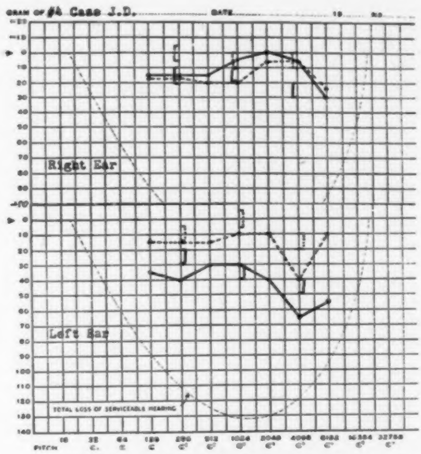
will be reported in the near future. In Group I there are nine cases; the majority of these were followed approximately

six months. Of these, six had a nasopharyngeal tumor. Of the remaining three patients, one had a carcinoma of the



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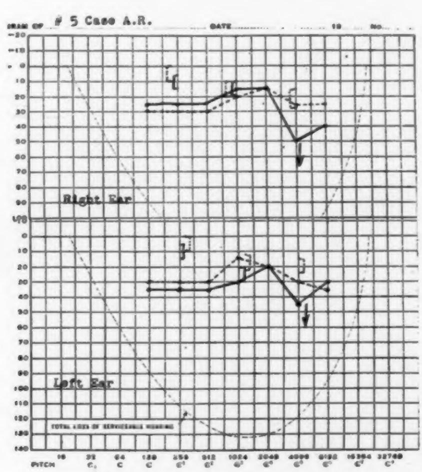

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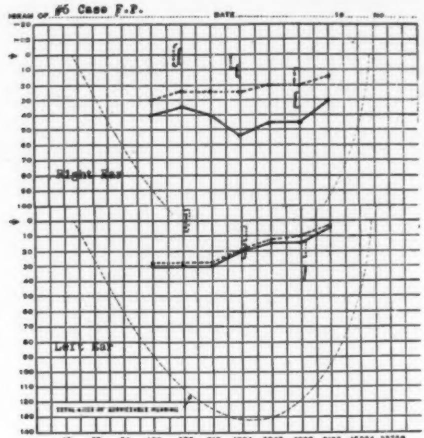
right tonsil with metastases to the right cervical lymph nodes; the second case had a carcinoma of the right antrum, and

the third had a basal cell epithelioma of the skin of the left preauricular region. The youngest of this group was age 17



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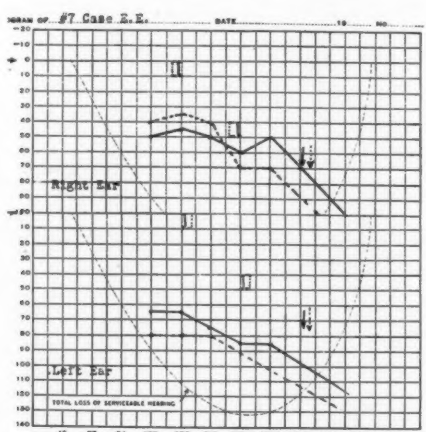

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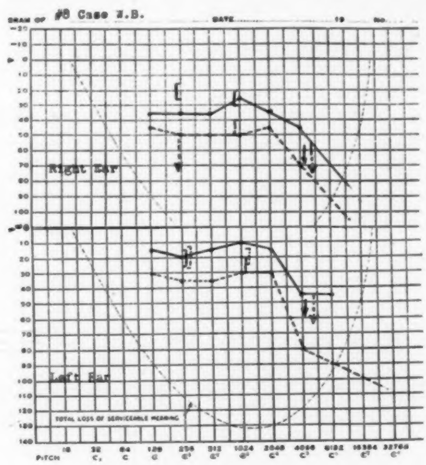
years and the oldest was age 61 years. Of the nine patients, two died recently. The remaining seven are living. Of these,

six are still able to be up and about. One patient at present is in the terminal stage of the disease.

TECHNIQUE OF ROENTGENTHERAPY.

In all these cases diagnosis was established by microscopic examination of the lesion and high voltage Roentgentherapy has been used. The technical factors are 200 KV, 1 mm. or 2 mm. Cu./1 mm. Al. and 50 or 80 cm. anode-skin distance. The quantity varied from 100 to 250 Roentgens per sitting, repeated at daily intervals, alternating over two or



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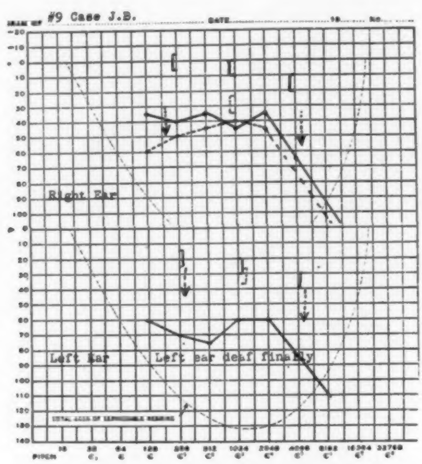
more areas. The total dosage varied between 2,000-4,000 Roentgens.

These patients were followed in otolaryngology and radiotherapy departments at frequent intervals. Audiograms were made before Roentgentherapy was applied; and repeated tests were made thereafter during and after the completion of the course of radiation. In the majority of the cases the readings were taken at weekly, then bimonthly, and, finally, at monthly intervals. Prior to radiation, audiograms were repeated so as to eliminate any possible error, and to get the patients accustomed to the routine procedure.

RESULTS.

The majority of these patients had more than 10 audiometric readings. For the sake of simplicity, it would seem sufficient to record the readings prior to the treatments and compare with the last audiograms in each case. These two readings are shown in graph form on individual hearing test sheets.

There were three cases (see Cases 7, 8, 9, Table I) which were afflicted with a far advanced neoplasm associated with anemia and inanition. Despite the initial regression of the growth following Roentgentherapy, their general condition



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became progressively worse. A series of audiogram readings disclosed marked diminution of acuity of hearing. In the remaining six cases the audiometric findings showed a simultaneous improvement in the air and bone conduction. In five of the patients there was a gain in acuity of 5 to 10 dcb.

DISCUSSION.

It is of interest to note that all of our patients received intensive radiation for the treatment of a neoplasm near the auditory system. This provided an unique opportunity to study the effect of large quantities of short-wave X-rays

on the acuity of hearing; whereas the investigations made by Sobotky, Jarvis and McCoy apply to irradiation in relatively smaller doses.

The improvement noted in six of our cases needs further comment. It is reasonable to assume that the primary site of the tumor may have a bearing on the impairment of the hearing. A tumor so located, or so extensive as to occlude the Eustachian tube, might have interfered with the function of the ear. Following a course of radiotherapy the tumor decreased in size or disappeared completely. Thus, by relieving the cause of compression against the Eustachian orifice, a notable improvement took place in the air conduction readings. In other instances, acuity of hearing varied in proportion with the general well-being of the patient during the course of radiotherapy. The hearing was at times impaired when the patient complained of "radiation sickness," or general weakness, but improvement took place soon after the cessation of these complaints.

Of the nine cases, only one patient had tinnitus (Case 1, R. G.), associated with an extensive nasopharyngeal cylindroma. It is only four months since the course of intensive radiation was completed. The tumor has regressed in size, but the patient obtained no relief of tinnitus.

Two of the nine cases received radiotherapy only on one side. One case (Case 8, W. B.) had a far advanced carcinoma of the right tonsil, with metastases to the cervical nodes. The area was irradiated through the right temporal bone posteriorly, and through the anterior aspect of the neck and right mandible. The left auditory region was in no way included in the field of radiation. The audiometric readings of this case are inconclusive, as the patient went down hill rapidly and succumbed to the disease. The second case (Case 5, A. R.) received intensive Roentgentherapy over the left ear for a basal cell epithelioma of the preauricular skin. The audiographic findings showed (see hearing sheet) more improvement on the treated side as compared with the untreated one.

In six cases the improvement of the air conduction was accompanied by improvement of bone conduction, as shown on the accompanying audiograms. It is as yet too early to evaluate the significance of this observation. However, it is reasonable to assume that intensive radiation might have a

direct effect on the nervous mechanism of the aural system. A similar investigation on cases with partial or complete nerve deafness might throw further light on this most interesting subject.

Of the nine patients, two died recently. The remaining seven are under observation. The majority of these patients have been followed on an average period of four months after the course of Roentgentherapy. Up to the present time, the acuity of hearing of six cases remains improved.

These investigations, of which the above report is but a preliminary statement, lead us to believe that the effect of high voltage Roentgen rays on the auditory system may have therapeutic significance in selected cases.

SUMMARY.

1. A preliminary report of nine cases with neoplasm in the nasopharynx or near the region of the ear is made. In treating the primary lesion the ears had to be included in the field of intensive radiation.

2. Six cases showed improvement in hearing. Three cases with rapid decline in general health showed marked decrease in acuity.

3. The acuity of hearing fluctuated with the general health and condition of the patient.

4. The Roentgen rays on the auditory system may have a therapeutic value in selected cases.

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SYMPOSIUM.

THE NEURAL MECHANISM OF HEARING.

V.—ETIOLOGICAL AND CLINICAL TYPES OF SO-CALLED "NERVE DEAFNESS."

"NERVE DEAFNESS" OF KNOWN PATHOLOGY OR ETIOLOGY.

INFLUENCE OF SOME TOXIC SUBSTANCES ON THE INNER EAR.*†

DR. ELLISON L. ROSS and DR. LUDWIG G. LEDERER, Chicago.

Toxic substances are responsible for a large proportion of the injuries that a living body receives. These substances may come from derangements of metabolism, from bacteria of disease or from poisons introduced into the body. The VIIIth nerve, as well as other anatomical structures, is subject to these destructive compounds.

In 1908, Van Rossem[1] showed that cocain applied in the middle ear produced anesthesia of the vestibular apparatus. Recently[2,3] it has been shown that a number of compounds pass from the middle ear to the inner one. It is suggestive that most soluble substances pass from the middle ear to the inner ear.

In our previous work[2,3] most of the data was on the immediate effects of substances put into the middle ear. In this paper observations will be given on the effects appearing over a period of one to three weeks.

EXPERIMENTAL WORK.

Dogs were used throughout the entire experimental work. In most instances the dog was first subjected to a control rotation.

All rotations were performed in the horizontal plane, allowing 30 turns in each direction, followed by an abrupt stop.

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The ensuing nystagmus was timed in seconds. Three rotations in each direction were averaged and figures stated in the appropriate table.

The following substances were used:

1. Diphtheria Toxin, 100, 50, and 25 per cent solutions—16 dogs. All dilutions of the toxin were made in 0.9 per cent sodium chloride solution.

2. Arsphenamine (salvarsan) in 1.0, 0.5, 0.1 per cent solutions—15 dogs. (In each instance the sodium salt was made according to the prescribed technique.)

3. Neoarsphenamine (neosalvarsan) in 1.0 and 0.5 per cent solution—9 dogs.

4. Salyrgan, 5.0 and 1.0 per cent solutions—5 dogs.

5. Quinine Bisulphate, 5 and 10 per cent solutions—7 dogs.

6. Phenol, 5.0 and 1.0 per cent solutions—7 dogs.

7. Methyl Alcohol, 10 per cent solutions—3 dogs.

The amount of the various substances injected into the middle ear cavity, according to the technique described in a previous paper,[3] was 1 c.cm. of fluid.

After the injection the animal was kept under ether anesthesia for a period of one hour to allow for the absorption of the drug. The head was maintained in such a position that the middle ear would not be emptied by way of either the Eustachian tube or the external canal.

Postinjection rotations were made in most instances at frequent intervals. The animals were kept for 6-25 days after the injection unless they died from toxemia.

Dogs showing a loss of vestibular function as evidenced by rotation were in most cases subjected to caloric tests. At the end of the period of observation the animals were anesthetized and perfused, first, with normal salt solution, and second, with a solution of 4 per cent formaldehyde and 1 per cent formic acid. This produced perfect and immediate fixation of the tissues of the head. The inner ears were prepared by the usual methods for microscopic study.

In a previous publication[3] some experiments were made on the effect of diphtheria toxin placed in the middle ear of dogs. Observations were made of the disturbance of equilibrium manifested by the position of the body, the gait, and spontaneous nystagmus. These observations were chiefly made immediately following the administration of the toxin and did not extend beyond several hours. It was thought to be of interest to make similar observations on the effect of diphtheria toxin over a longer period of time and also to use various concentrations of the toxin.

Seven animals were injected in the right middle ear with full strength of commercial diphtheria toxin. Observations on the posture, gait, and spontaneous nystagmus were made daily. Every two to four days rotation tests were made in a horizontal plane on a mechanical rotator, with resulting nystagmus observed in seconds of duration. Three or more rotations were made in each direction. The animals were kept for approximately 10 to 15 days. Three died after a shorter period. The temporal bones were preserved for microscopic examination. Animal No. 104 was a typical animal of this group. In order to abbreviate this report, the detailed record of this animal will be the only one presented.

PROTOCOL DOG NO. 104—DIPHTHERIA TOXIN.

Dog No. 5 of Diphtheria Series.—Black and white dog with black spots on back.

	Rotation	
	To Right	To Left
Dec. 1, 1936.—Control Rotation.	22.8	21.8
	21.4	19.4
	21.0	20.6
	Av. 21.7	Av. 20.6

Dec. 1.—Dog was injected—right ear—same technique as for other dogs. No nystagmus followed.

Dec. 1.—Five hours after injection.	18.8	20.4
	18.8	20.2
	19.0	20.0
	18.9	20.2
Dec. 2.	16.6	18.6
	15.8	17.0
	15.8	17.2
	16.1	17.6

Dec. 3.	18.4 19.4 18.2	Signs of rt. labyrinth deficiency developing	13.6 13.4 13.6
	18.7		13.5
Dec. 4.	22.5 22.6 22.8		13.4 12.2 12.0
	22.6		12.5
Dec. 6.	25.0 25.8 27.2		10.6 11.0 11.4
	26.0		11.0
Dec. 8.—Before injection.	16.2 17.0 17.4		8.0 8.2 7.4
	16.9		7.9
Dec. 8—Dog was injected into left middle ear same technique as for right. Ten minutes after emerging from anesthesia, dog had a nystagmus to the left, which remained active for two hours.			
Dec. 9.	16.2 16.8 17.4		8.8 8.2 7.0
	16.8		8.0
Dec. 11.	15.0 15.2 16.0		9.2 9.0 8.6
	15.4		8.9
Dec. 13.—Marked nystagmus while it lasted.	7.0 5.6 4.2		2.0 3.6 0.0
	5.6		1.9
Dec. 15.	4.2 4.0 3.0		9.2 5.4 4.2
	3.7		6.3

Dec. 15.—Calorics done—negative on both sides after inserting approximately 4 oz. ice water. Dog etherized perfused and killed with saline and 10 per cent formalin and 1 per cent formic acid. Tissues fixed in 10 per cent formalin and designated as Dog No. 104.

Five dogs were similarly treated with 50 per cent strength of diphtheria toxin. Four of them were treated with 25 per cent strength of toxin.

The essential data on the effect of the diphtheria toxin placed in the middle ear is presented in Table I.

TABLE I.

EFFECT OF DIPHTHERIA TOXIN.

Dog. No.	Days of Observation.	Concentration.	Duration of (a) Spont. Nystagmus After First Day.	Nystagmus After Rotation (b) to Right	Left	Dead Labyrinth Proved By Caloric.
2 (101)	11	100%	None	25.2 sec.	3.5 sec.	
3 (100)	6	100%	6th P.O. Day—3 days	22.3 sec.	14.3 sec.	
4 (102)	9	100%	None	38.6 sec.	8.8 sec.	
5 (104)	15	100%	None	21.8 sec.	10.5 sec.	Positive
6	12	100%	None	15.4 sec.	15.7 sec.	
7 (108)	15	100%	5th P.O. Day—10 Days	22.2 sec.	10.2 sec.	Positive
8 (107)	12	100%	None	18.5 sec.	7.8 sec.	
9	4	50%	None	17.0 sec.	12.5 sec.	
10 (120)	20	50%	None	10.9 sec.	5.7 sec.	Positive
11 (121)	10	50%	9th P.O. Day—1 Day	23.9 sec.	8.5 sec.	Positive
12 (125)	11	50%	None	12.3 sec.	10.0 sec.	Positive
13 (126)	10	50%	10th P.O. Day—2 Days	21.5 sec.	7.1 sec.	Positive
14 (127)	14	25%	None	25.5 sec.	10.5 sec.	Positive
15 (128)	14	25%	7th P.O. Day—1 Day	15.1 sec.	6.6 sec.	Positive
16 (134)	13	25%	None	18.0 sec.	8.8 sec.	Positive
17 (135)	13	25%	None	18.2 sec.	10.1 sec.	Positive

(a)—Nystagmus to the uninjected side.
(b)—Average of the last three days of rotation.

For the group of seven animals receiving full strength of diphtheria toxin, the rotation toward the side injected produced a very much longer period of nystagmus than that in the opposite direction except in the case of animal No. 6. In all of the group, with this exception, there was the characteristic turn of the head to the side of the injected ear and a tendency to fall in the same direction. Caloric tests proving a dead labyrinth were performed in only two of this group. It was quite clear that the labyrinths were destroyed in six of the seven animals. It is thought that the failure in the one was due to faulty injection of the middle ear.

Of the five animals receiving 50 per cent strength of the diphtheria toxin, all but one showed the decided difference in periods of nystagmus resulting from rotation in the two directions, a characteristic of a dead labyrinth in the side injected. In No. 125, which showed a small difference in the periods of nystagmus, a caloric test proved the presence of a dead labyrinth. In all of the subjects there developed the posture and gait characteristic of a dead labyrinth in the side injected. It must be concluded that 50 per cent strength of diphtheria

toxin in the middle ear is capable of destroying the function of the vestibular apparatus.

Four animals receiving 25 per cent strength of diphtheria toxin also gave the characteristic difference in nystagmus resulting from the rotation in opposite directions. In all four animals a caloric test proved the presence of a dead labyrinth. The posture and gait characteristic of a dead labyrinth were present in all the subjects. Evidently 1 cc. of 25 per cent strength of diphtheria toxin placed in the middle ear will destroy the function of the labyrinth on that side.

It is common knowledge that arsphenamine when used clinically is capable of injuring the optic nerve. Some reports have been made[4] of cases of deafness resulting from administration of this drug. Therefore, it was considered advisable to investigate the effect of this drug on the vestibular apparatus.

Three concentrations of arsphenamine were used in the middle ears of dogs in a manner similar to that employed with diphtheria toxin. Five received a 1 per cent solution, four a 0.5 per cent solution, and four a 0.1 per cent solution. The following is a detailed record of No. 111, which is typical of the series:

PROTOCOL DOG NO. 111—1 PER CENT ARSPHENAMINE.

Dog No. 1.—Gray dog with tan spot between eyes.

	Rotation To Right	Rotation To Left
Dec. 26, 1936.—Control Rotation.	17.2	15.0
	15.8	15.8
	15.0	14.8
	Av. 16.0	Av. 15.2
Dec. 26.—This dog's right ear was injected with 1 cc. of 1 per cent arsphenamine. Anesthesia for absorption for 1 hour followed. Dog had a nystagmus to the left for 20 minutes after coming out of anesthesia.		
Dec. 27.—This dog had an active nystagmus to the left so violent in character that rotation results could not be obtained.		
Dec. 28.—There is still present such violent nystagmus that no rotation can be made.		
Dec. 29.—Nystagmus to left present all the time.		7.4
		9.2
		7.4
		8.0

Dec. 30.—Nystagmus to left still present.		7.0 7.8 7.0
		7.3
Jan. 1, 1937.	15.8 17.6 17.4	7.0 7.0 7.4
	16.9	7.0
Jan. 3	18.0 16.8 17.2	6.0 5.6 5.8
	17.3	5.8
Jan. 6.	17.4 17.8 16.8	6.0 5.8 6.2
	17.3	6.0
Jan. 9.	16.2 19.0 16.4	6.0 5.8 5.4
	17.2	5.7
Jan. 13.	14.2 15.6 16.2	6.2 6.0 5.4
	15.3	5.9
Jan. 18.	13.8 14.2 14.0	7.0 7.2 7.0
	14.0	7.1

Jan. 20.—This dog is now 25 days postinjection. Calorics were done. **Rt. ear negative**—and the drum seemed perforated because water emerged through the nose. **Left ear**—caloric gave irregular results—could not be accurately timed, but there seemed to be a suggestion of nystagmus to the right.

This dog was perfused and killed with 10 per cent formalin mixture according to our regular technique.

In Table II the essential data on the three series of animals receiving arsphenamine are presented.

TABLE II.

EFFECT OF ARSPHENAMINE (SALVARSAN) NO. 606.

Dog. No.	Days of Observation.	Concentration.	Duration of (a) Spont. Nystagmus After First Day.	Nystagmus After Rotation (b) to Right	Left	Dead Labyrinth Proved By Caloric.
1 (111)	23	1.0%	4 days postinjection	15.5 sec.	6.2 sec.	Positive
2 (113)	21	1.0%	3 days postinjection	15.8 sec.	5.8 sec.	Positive
4 (114)	20	1.0%	3 days postinjection	13.1 sec.	6.6 sec.	Positive
5 (115)	23	1.0%	4 days postinjection	13.3 sec.	6.3 sec.	Positive
6 (116)	23	1.0%	4 days postinjection	9.4 sec.	5.2 sec.	Positive
7	20	0.5%	2 days postinjection	13.2 sec.	13.1 sec.	
8 (112)	20	0.5%	2 days postinjection	11.4 sec.	6.1 sec.	Positive
9 (119)	18	0.5%	1 day postinjection	14.0 sec.	9.4 sec.	Positive
10 (117)	9	0.5%	1 day postinjection	17.2 sec.	7.4 sec.	
11	12	0.1%	None observed	14.3 sec.	14.1 sec.	
12	12	0.1%	1 day postinjection	14.5 sec.	17.8 sec.	
13	10	0.1%	None observed	16.9 sec.	16.6 sec.	
14	10	0.1%	None observed	15.2 sec.	20.3 sec.	
15 (136)	3	1.0%	3 days postoperative	Sacrificed for study of acute		
16 (137)	4	1.0%	4 days postoperative	effects on cochlea.		

(a)—Nystagmus to the uninjected side.
(b)—Average of the last three days of rotation.

The animals receiving 1.0 per cent arsphenamine showed unmistakable signs of a dead labyrinth on the side in which the compound was applied. Three to four days of spontaneous nystagmus away from the injected ear occurred. Rotation toward the side of the injected ear always produced a decidedly longer period of nystagmus than that resulting from rotation in the opposite direction. The caloric test in every case indicated a dead labyrinth on the side injected.

Four subjects receiving 0.5 per cent strength of arsphenamine were less disturbed and showed less constant results. Spontaneous nystagmus away from the injected side lasted but one to two days. In animal No. 7 there was no difference in the periods of nystagmus. In the other three there was the characteristic difference in the periods of nystagmus. In two of this series caloric tests were made which in each case indicated a dead labyrinth. The results suggest that 0.5 per cent strength of arsphenamine is capable of destroying the labyrinth in some cases only.

Another series of four received .1 per cent concentration of arsphenamine. Only one of these animals showed a spontaneous nystagmus away from the injected side and that for a

period of one day only. The differences in the duration of nystagmus resulting from rotation in the two directions did not indicate a loss of function in the injected side, but in two cases suggested a stimulation.

The data in Table II indicates that approximately 1 cc. of 1 per cent arsphenamine in the middle ear under the conditions of the experiment will uniformly destroy the function of the vestibular apparatus. Arsphenamine in .5 per cent concentration may under most favorable conditions destroy the labyrinth. Arsphenamine in .1 per cent concentration in the middle ear proved harmless to the vestibular mechanism.

Since arsphenamine had given toxic effects on the vestibular nerve, it was thought advisable to make similar observations after neoarsphenamine. Table III contains the data obtained using this compound.

TABLE III.

EFFECT OF NEOARSPHENAMINE—NEOSALVARSAN.

Dog. No.	Days of Observation.	Concentration.	Duration of (a) Spont. Nystagmus After First Day.	Nystagmus After Rotation (b) to Right	Left	Dead Labyrinth Proved By Caloric.
1 (122)	11	1.0%	2 days postinjection	17.4 sec.	8.4 sec.	Positive
2 (123)	11	1.0%	2 days postinjection	21.4 sec.	9.4 sec.	Positive
3 (124)	11	1.0%	2 days postinjection	20.1 sec.	10.1 sec.	Positive
4	13	0.5%	1 day postinjection	15.2 sec.	16.5 sec.	
5 (118)	14	0.5%	1 day postinjection	14.0 sec.	7.5 sec.	
6	18	0.5%	None observed	10.8 sec.	9.6 sec.	
7	14	0.5%	3 days postinjection	11.2 sec.	11.9 sec.	
8	13	0.5%	3 days postinjection	11.8 sec.	23.6 sec.	
9 (129)	12	0.5%	3 days postinjection	15.3 sec.	7.3 sec.	Positive

(a)—Nystagmus to the uninjected side.
(b)—Average of the last three days of rotation.

Three animals were given 1 per cent neoarsphenamine in the usual way. A spontaneous nystagmus away from the injected side was present for two days. The rotation to the injected side was approximately twice as long as that resulting from the rotation in the other direction. The position of body and the gait were characteristic of a dead labyrinth on the injected side. Caloric tests in each case indicated a dead labyrinth on the injected side. There is no doubt that in these three animals the vestibular function was destroyed by 1 cc. of 1 per cent neoarsphenamine placed in the middle ear.

A solution of .5 per cent neoarsphenamine was administered in the usual way to six dogs. Three had spontaneous nystagmus for three days following the injection. Two had it for one day and one did not develop any spontaneous nystagmus. Only two of the animals showed a difference in post-rotatory nystagmus characteristic of a dead labyrinth. It is quite apparent that a 0.5 per cent strength of neoarsphenamine may be expected to give irregular results in the destruction of labyrinthine functions due to its administration into the middle ear.

The use of mercury in the treatment of disease has quite frequently affected the nervous system. This led us to try the effect of mercury on the vestibular apparatus. Salyrgan was chosen because it is soluble and relatively nonirritating. A 5 per cent and a 1 per cent solution were employed. Table IV presents the data on this subject.

TABLE IV.
EFFECT OF SALYRGAN (ORGANIC MERCURY).

Dog. No.	Days of Observation.	Concentration.	Duration of (a) Spont. Nystagmus After First Day.	Nystagmus After Rotation (b) to Right	Left	Dead Labyrinth Proved By Caloric.
1 (130)	11	5.0%	2 days postinjection	12.6 sec.	9.0 sec.	Twice pos.
2 (131)	11	5.0%	2 days postinjection	26.3 sec.	9.0 sec.	Twice pos.
3 (132)	11	5.0%	2 days postinjection	16.2 sec.	8.9 sec.	Twice pos.
4	17	1.0%	None observed	11.2 sec.	15.7 sec.	None pfd.
5	17	1.0%	None observed	15.3 sec.	22.4 sec.	None pfd.

(a)—Nystagmus to the uninjected side.
(b)—Average of the last three days of rotation.

The three subjects receiving 5 per cent solution of salyrgan had spontaneous nystagmus away from the injected side for two days. The nystagmus following rotations in the two directions indicated a dead labyrinth on one side. In each of the three animals two caloric tests indicated a dead labyrinth on the side of injection.

Two animals were given 1 per cent salyrgan in the usual way. No spontaneous nystagmus was observed. Rotation toward the injected side gave a shorter nystagmus than in the other direction, which indicated a stimulation by the drug rather than a depression. It may be concluded that mercury

in the form of salyrgan is capable of destroying the vestibular function, but this must be in a concentration over 1 per cent and not necessarily more than 5 per cent.

In a previous article[3] there was reported the immediate effects of 5 per cent quinine bisulphate injected into the middle ear. The results indicated the drug to be an immediate depressant upon the vestibular organ. There still remained unanswered the question as to whether the depression was permanent or temporary. Table V contains the essential records resulting from the use of quinine.

Injected into ME cavity p. 556

TABLE V.

EFFECT OF QUININE BISULPHATE.

Dog. No.	Days of Observation.	Concentration.	Duration of (a) Spont. Nystagmus After First Day.	Nystagmus After Rotation (b) to Right	Left
1	10	5.0%	3 hours postinjection	12.4 sec.	13.1 sec.
2	13	5.0%	3 hours postinjection	20.1 sec.	16.6 sec.
3 (103)	19	5.0%	12 hours postinjection	12.2 sec.	13.9 sec.
4 (106)	25	5.0%	12 hours postinjection	16.8 sec.	14.9 sec.
5 (105)	21	5.0%	12 hours postinjection	11.1 sec.	20.3 sec.
6 (109)	10	10.0%	2 hours postinjection	15.5 sec.	15.3 sec.
7 (110)	11	10.0%	2 hrs. 20 min. postinj.	14.9 sec.	11.6 sec.

(a)—Nystagmus to the uninjected side.
(b)—Average of the last three days of rotation.

Five animals were given 5 per cent quinine bisulphate. A spontaneous nystagmus following the injection was present in all cases, but not beyond the one day. Postrotatory nystagmus characteristic of one dead labyrinth was present in none of the five cases. Caloric tests did not indicate the presence of a dead labyrinth. The two animals which received 10 per cent solution of quinine bisulphate developed very short periods of spontaneous nystagmus. The postrotatory nystagmus was not sufficiently different for the two directions to conclude the presence of a dead labyrinth. The results from the caloric tests were uncertain. These tests did indicate that quinine bisulphate acting directly upon the vestibular nerve endings acted as an anesthetic rather than as a destructive agent.

Carbolic acid is always listed among the common toxic compounds and is a common constituent of ear medication.

A short series of animals subjected to the influence of this drug was observed. The data obtained are given in Table VI.

TABLE VI.
EFFECT OF CARBOLIC ACID.

Dog. No.	Days of Observation.	Concentration.	Duration of (a) Spont. Nystagmus After First Day.	Last 3 Days of Rotation.	Nystagmus After Rotation (b) to Right	Left	Caloric Test of Labyrinth
1	13	5.0%	1 day postinjection	11-13*	17.9 sec.*	17.1 sec.*	2 alive
2	13	5.0%	1 day postinjection	11-13*	14.1 sec.*	12.8 sec.*	3 alive
3	13	5.0%	1 day postinjection	11-13*	16.6 sec.*	23.5 sec.*	3 alive
4	7	5.0%	7 hours to left	7 only	27.5 sec	20.5 sec.	alive
5	7	5.0%	7 hours to left	7 only	10.3 sec.	10.1 sec.	alive
6	10	1.0%	None observed	4-7-10	16.4 sec.	16.9 sec.	Nono perfmd.
7	10	1.0%	None observed	4-7-10	10.5 sec.	11.6 sec.	None perfmd.

(a)—Nystagmus to the uninjected side.
(b)—Average of the last three days of rotation.
*Last two days of rotation.

Five subjects were given 5 per cent phenol in normal salt solution. Spontaneous nystagmus away from the injected ear was present during the day of injection. At the end of 13 days, rotation in the two directions produced no significant difference in the resulting nystagmus for three of the dogs. The remaining animals gave similar results at the end of seven days. Caloric tests indicated both labyrinths alive. Two animals were given 1 per cent phenol. There resulted no evidence of either temporary or permanent vestibular disturbance. It is apparent that carbolic acid in the concentration of 5 per cent is capable of producing an anesthesia or a temporary depression of the vestibular organ on the side injected.

It has been reported[3] that methyl alcohol in a concentration of 50 per cent placed in the middle ear produced immediate depression of the vestibular function on that side. Methyl alcohol is one of the common poisons of the optic nerve. Many cases of blindness with optic nerve degenerations have resulted from drinking or inhaling methyl alcohol. It seems impossible that the concentration of methyl alcohol in these cases could have been anywhere nearly as great as 10 per cent in the nerve ending. A 10 per cent solution of

methyl alcohol was used in the middle ear of three animals. The results of this series of tests are given in Table VII.

TABLE VII.

EFFECT OF METHYL ALCOHOL.

Dog No.	Days of Observation.	Concentration.	Duration of (a) Spont. Nystagmus After First Day.	Nystagmus After Rotation (b) to Right	Left
1	14	10.0%	None observed	13.4 sec.	13.8 sec.
2	14	10.0%	None observed	12.0 sec.	9.3 sec.
3	14	10.0%	None observed	11.8 sec.	11.9 sec.

(a)—Nystagmus to the uninjected side.
(b)—Average of the last three days of rotation.

No spontaneous nystagmus was observed at any time. At the end of 14 days the rotation tests indicated that the labyrinth on the injected side was alive and in two out of the three cases as active as the other.

It is known that diphtheria toxin, arsphenamine, and neoarsphenamine are strong irritants. It was a question whether these substances might not act by necrosis, rather than by changing tissue cell metabolism. To test the necrotic possibilities of these poisons, small balls of cotton saturated with each substance were placed for an hour under the lower lids of anesthetized dogs. Observations were made daily. Two animals were tested with 100 per cent strength of diphtheria toxin. There resulted considerable redness and swelling of the conjunctiva. The cornea became opaque, and remained so for over five days. Mercurochrome was applied repeatedly which showed no break in the surfaces. Two animals were subjected to 1.0 per cent arsphenamine in the same way. The inflammatory changes were somewhat greater and there was a very slight erosion of the conjunctiva. Three dogs were given neoarsphenamine of 1.0 per cent in the eye. There was marked reddening and swelling. The cornea became opaque. There was no sign of erosion when tested with mercurochrome. Therefore, it is not likely that the effects of diphtheria toxin, arsphenamine, or neoarsphenamine were due to an erosive action.

The temporal bones of some of the animals which received diphtheria toxin of 100 per cent strength and arsphenamine

of 1.0 per cent strength were examined microscopically. They were prepared in the usual way, and stained with hemotoxylineosine and also by Weigert's method.*

Fig. 1 is a photomicrograph of a section of the organ of Corti which had been treated with diphtheria toxin of 100 per cent strength. The cells of limbus laminae spiralis were indis-

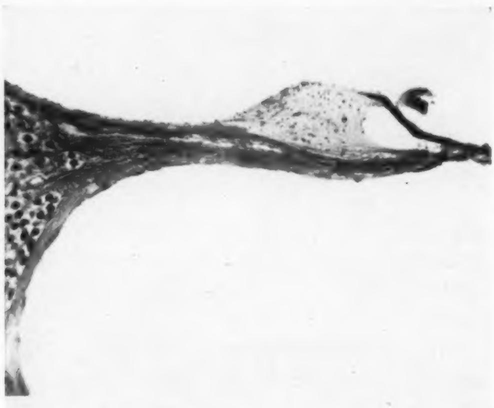

Fig. 1.

Fig. 2.

tinct, pale, and shrunken. The membrana tectoria was much thinner and had lost its fibrils. The pillar and hair cells were indistinct and irregular. The tunnel of Corti was obliterated. The nerve fibres leading to the organ of Corti seemed smooth and compact.

*Much assistance and some of the slides were provided by Dr. Arthur Weil of the Department of Neuro-pathology.

Fig. 2 shows a section of an organ of Corti which was subjected to arsphenamine of 1.0 per cent. The changes present are of about the same type and degree as those formed after diphtheria toxin.

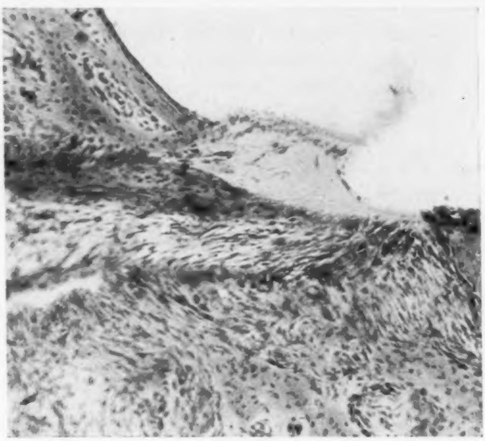

Fig. 3.

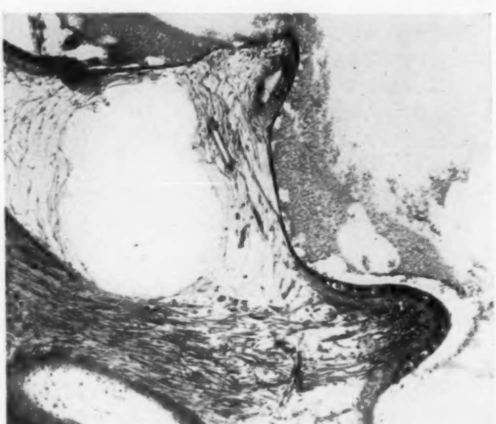

Fig. 4.

Fig. 3 shows a photomicrograph of another organ of Corti which had been changed by the same strength of arsphenamine. Here there was less atrophy of cells, such as those

of the limbus, membrana tectoria, and the pillar and hair cells. However, the nerve fibres to the organ of Corti could be picked out with difficulty amid the large mass of fibrous tissue in the region. The scala tympani was filled with organizing tissue (fibrous tissue, cartilage, and possibly bone).

Fig. 4 is a photomicrograph of the ampulla which was treated with arsphenamine. The greatest change from normal is the swelling of the outer layer of cells and the disappearance of the cupula.

LITERATURE.

Wittmaack[5] removed the bullae of cats and injected various living bacteria through the round window. He studied the inner ear changes.

Blau[6] carried on the same type of work as Wittmaack. Labyrinthitis resulted and varying degrees of destruction of the inner ear were noted. Wittmaack has recently reported severe osmotic changes in the inner ear following the injection of methanamine in the middle ear of guinea pigs.[7]

We have been unable to find in the literature records of changes in the inner ear produced by the presence of the herewith reported toxic substances in the middle ear. The clinical conditions which may be similar to it are diseases that are more or less primary in the middle ear, such as otitis media, diphtheria, and scarlet fever. The literature on this phase of the subject may be found in our previous article.[3]

SUMMARY AND CONCLUSIONS.

Diphtheria toxin of 100, 50, and 25 per cent strength of Parke-Davis preparation with M.L.D. of .0042 cc. was injected into the middle ears of dogs and kept there for one hour.

The strongest concentration destroyed the vestibular functions on the side injected and the microscopic examination revealed much damage done to the cochlear organ.

Weaker concentrations of the diphtheria toxin destroyed the vestibular function in most cases.

Arsphenamine and neoarsphenamine used in the same manner as the diphtheria toxin destroyed vestibular function uni-

formly when in a concentration of 1 per cent. Microscopic examinations of the ears nine to 23 days following the injection of arsphenamine showed marked injury to the nerve cells and tissue cells of both the ampullae and organ of Corti.

Concentrations of the arsphenamines of 0.5 per cent destroyed the vestibular function in the majority of animals, while 0.1 per cent had no permanent effect.

Salyrgan in 5 per cent strength in the middle ear destroyed the vestibular function. When used in 1 per cent concentration, no effect was observed.

Quinine of 10 per cent strength only temporarily depressed the vestibular function. The same was true with 5.0 per cent phenol. Apparently both act as anesthetics.

Methyl alcohol in 10 per cent strength in the middle ear had no effect on equilibrium.

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Northwestern University.

SYMPOSIUM.

THE NEURAL MECHANISM OF HEARING.

V.—ETIOLOGICAL AND CLINICAL TYPES OF SO-CALLED "NERVE DEAFNESS."

(b)—"NERVE DEAFNESS" OF KNOWN PATHOLOGY OR ETIOLOGY.

NERVE DEAFNESS FROM SYPHILIS.*†

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A review of the extensive literature concerning syphilis of the VIIIth cranial nerve shows that the auditory defects most commonly recorded are: *1.* Loss of acuity for high tones and a lowered upper tone limit; *2.* loss of acuity for all tones by air conduction and impaired bone conduction; *3.* shortened bone conduction associated with "normal" hearing by air conduction. Since, with exception of the last, these defects constitute the classical signs of "nerve" deafness, it is important to determine whether or not these particular hearing defects are found more frequently in syphilitic patients, and if there are any forms of "nerve" deafness which may be regarded as pathognomonic of syphilis.

AUDITORY DEFECTS INVOLVING HIGH TONES OR ACCOMPANIED BY IMPAIRMENT FOR LOW AND MIDDLE TONES.

The first investigator to utilize the audiometer in order to determine whether loss of acuity for high tones is more frequent in syphilitics was Bunch, who, in 1931, published the results of his study based on material collected in the Otological Research Laboratory of the Johns Hopkins University. Bunch found that syphilitic patients, selected because they did not complain of any hearing disturbance, had on the average the same amount of high tone loss as other hospitalized patients of their particular age groups.

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†From Division of Public Health Methods, National Institute of Health, U. S. Public Health Service.

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A somewhat different method of approach to the problem was later employed by me in the study of additional data collected in the Otological Research Laboratory. The material utilized included all the hospital patients tested up to that time as part of the routine of the laboratory. Statistical analysis of data from this group, which consisted of a random sample of hospital patients, showed that high tone loss and other forms of so-called inner ear deafness are no more frequent in luetic than in non-luetic patients of the same age and sex. From these studies it can be assumed that if one examines a group of syphilitic patients selected at random, there will be among them as many persons with the several forms of "nerve" deafness as is found in the general hospital population.

These studies also furnished information regarding Drury's statement that in cases with a dip at 4,096 d.v., the existence of a luetic taint might be regarded as probable. Thus, it was observed, in a large sample of hospital patients, that the 4,096 dip was not present any more frequently in syphilitics than in nonsyphilitics.

DISPROPORTIONATE SHORTENING OF THE BONE CONDUCTION.

A shortened bone conduction time—usually measured with a 435 or 512 d.v. tuning fork—associated with good hearing by air conduction rather generally has been regarded as pathognomonic of syphilis since Beck's observations in 1913. A critical review of the literature shows, however, that the reported incidence of this symptom in syphilitic patients varies from 2 to 95 per cent. This enormous variation is essentially due to the lack of a precise definition of what constitutes shortened bone conduction. In my studies on the subject, based on data collected in the Otological Research Laboratory, bone conduction measured with a 512 d.v. fork was considered shortened when the time the fork was perceived by the subject was less than half that of the examiner. A comparison of the bone conduction time in almost 1,000 hospital patients whose hearing by air conduction up to and including 1,024 d.v. was good showed that the incidence of disproportionate shortening of bone conduction in the syphilitic patients was substantially the same as that in nonsyphilitics. In fact, age appeared to be the only clinical find-

ing related to decrease in bone conduction acuity. The older patients had more often a shortened bone conduction time than the younger ones.

HEARING AND TYPE OF SYPHILIS.

Some evidence has been presented that the frequency of hearing impairment in syphilitic patients is associated with the type of syphilis affecting the individual. According to Alexander and to Krassnig, about 20 per cent of patients with latent syphilis acquire some form of "nerve" deafness. This is found in about 69 per cent of patients with central nervous system syphilis. Data collected by Weinstein and the author show that hearing impairment of the inner ear type is found about twice as frequently in patients with neurosyphilis as in patients with other forms of late syphilis.

Many observers have considered disturbance of function of the VIIIth nerve as evidence of central nervous system involvement in patients who had syphilis, or in whom that disease was suspected. Material which Weinstein and I have collected does not support this conclusion. The cerebral spinal fluid was examined in 106 patients with "nerve" deafness. Thirty of these had late syphilis and the spinal fluid was negative. In 64 with neurosyphilis, the spinal fluid was normal in 16 and abnormal in 48. Normal fluid was found in 11 of 12 with congenital syphilis. According to these findings, a diagnosis of neurosyphilis, based only on impaired function of the VIIIth nerve in a patient with syphilis, is not justified.

EFFECT OF ANTISYPHILITIC TREATMENT.

Otologists and syphilologists have given some attention to the effect of antisyphilitic treatment on the hearing. Statistical reports by Beck and by Lund, and experimental studies by Ferretti and others concerning the effect of arsphenamine and its various compounds indicate that the reported deleterious effects of these drugs have been exaggerated. Thus, there is reason to believe that patients with transitory impairment of hearing following an injection probably suffer from a form of Jarisch-Herxheimer reaction. "Nerve" deafness coming on one to two months following the inadequate treatment of early syphilis is considered to be not a toxic neuritis but

a neurorecurrence. As to the beneficial effects of treatment, Alexander states that in a group of syphilitic patients with impaired hearing which he kept under observation, 8 per cent regained normal hearing, 42 per cent improved, 48 per cent remained unchanged, and in 2 per cent the hearing became worse. Benario (quoted from Alexander) reported that in his cases of neurorecurrence the hearing was restored in 83 per cent while under treatment, and improved in another 8 per cent. He regards the age of the patient and the method of treatment as important. Krassnig observed no improvement of hearing in deaf or partially deaf tabetics and paretics.

In the study by Weinstein and myself, based on the hearing and clinical records of almost 300 patients with syphilis, it was observed that hearing impairment was just as frequent among the patients who had received adequate and intensive antisyphilitic treatment as among those who had been treated inadequately, or not at all. Sixty-one patients were reexamined otologically after an average interval of two years. During this period the hearing had not changed in 52; it had improved in four and became worse in five. The two patients showing definite and marked hearing improvement had early meningeal neurosyphilis (neurorecurrence); the two patients with greatest hearing decrease had late syphilis. From this study, it appears that there is no evidence of any relation between the amount of antisyphilitic treatment and the development of hearing defects. With the exception of deafness associated with early meningeal neusyphilis, treatment has no effect on hearing, either beneficial or detrimental.

SUMMARY.

The findings here reported indicate that:

1. Syphilis apparently does not cause "nerve" deafness more often than any other general systemic disease, nor is there any particular type of "nerve" deafness found more frequently in syphilitic patients.

2. When hearing impairment does occur it is found more frequently in persons with neurosyphilis.

3. Antisyphilitic treatment is apparently neither deleterious nor beneficial in the majority of cases.

Before closing, I wish to call to your attention the fact that the studies cited here, as well as others, are based upon patients with impaired hearing and with syphilis, but no evidence has been given that in the individual cases there is any causal relationship between the impairment and the disease.

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SYMPOSIUM.

THE NEURAL MECHANISM OF HEARING.

V.—ETIOLOGICAL AND CLINICAL TYPES OF SO-CALLED "NERVE DEAFNESS."

NERVE DEAFNESS FROM INFLAMMATORY LESIONS.*

DR. SAMUEL J. KOPETZKY, New York.

I am under no delusion, and I desire at the outset to free you of any impression that in this, my contribution to this symposium, I shall present anything which is new, or anything that I have personally evolved. Because it becomes necessary to be recorded in order to complete the symposium, I shall present what is accepted knowledge recorded in our literature and standard textbooks. I acknowledge freely having used the works of Wittmaack, Lange and Steingut, whose original works and observations in this field are otologic classics. Nor can I go into those fascinating histopathologic details to demonstrate how the tissue reactions to pyogenic infection, as it affects the bone-tissue surrounding the vital, living membranous labyrinth, and its nerve tracts to the brain, affects each cell strata to terminate and leave behind it, as a postinfectional state a varying degree of nerve deafness. But I shall endeavor to outline underlying pathologic principles which are generally applicable, and thus be enabled to better comprehend such lesions from the histopathologic standpoint, on the one hand, and on the other, perhaps, too, permit us a clearer understanding of their inherent clinical import.

Fundamentally, we come again to the primary consideration of the three types of bone with which we deal. The pneumatic, the diploic, and the eburnized, or sclerotic. The tender membranous labyrinth, encased in its capsule, is more generally prone to attack when it is lodged in a temporal bone which is sclerotic than when it is pneumatic. The diploic type of bone holds a middle place in factors which count toward

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frequency of labyrinthine involvement. It should be understood that inflammatory reactions in the labyrinth are only rarely primary involvements. Almost always they are secondary lesions. The exception is to be noted in thrombosis of the internal auditory vein, which, when infected, produces so rapid a change in the intralabyrinthine structures, and so extensive a disturbance by regression thrombosis, that the delicate nerve tissue succumbs, and if the patient's life is saved the extent of this nerve tissue loss results in varying degrees of a nerve type of deafness.

The pathways by which the labyrinth is affected, whether the primary lesion is in the tympanomastoid, the petrosal pyramid, or the meninges, are mainly three: through the blood stream, through the labyrinthine lymph channels, or through preformed anatomical pathways, particularly through the oval and the round-windows.

We can dismiss the hematogenic route, as seen in the anemias, leukemias, in the devitalizing diseases like nephritis and lues, because these have already been discussed in our symposium. One point only need be added. In the transmission of the infection along blood vessels, it is to be noted that Lange observed pus streaks leading from the middle ear to the endosteal capsular bone layer of the labyrinth, but these rarely followed preformed blood channels, but rather streaked along tangential blood channels which grow about an erosion process. The blood vessels involved appeared as new growths from the endosteal capsular layer.

The meningitic route, however, holds our attention for the moment because, from meningeal infections, particularly from epidemic meningoencephalitis, the lymph channels along the branches of the acoustic nerve, especially about the ramus cochlearis, may bring both bacteria and their products through the internal auditory meatus into the membranous labyrinth. Then, too, the direct communication of the perilymphatic space with the subarachnoidal space through the ductus aqueductus cochlea affords a passage whereby a suppurative meningitis can send its purulency to the membranous labyrinth. Naturally, because we are wont more often to face tympanomastoidal infections in our daily work, the tympanogenic route most often concerns us. Where the bone is pneumatic, and in the course of the development of a coalescing

mastoiditis, the cellular structure breaks down easily, there is less likelihood for the inflammatory reactions to set up their peculiar series of phenomena in the region of the round- and the oval-windows, as they perforce must do, where the bone is sclerotic and strongly resistant to pressure factors, and therefore more easily sets up chemical reactions induced by the closely confined purulent products in the tympanic cavity around the membranes of these windows, thus early starting reactions in the labyrinthine interior. A word as to the mechanics of this labyrinthine invasion. The phenomena is mostly observed about the foramen rotunda. The membrane itself is easily permeable. On its tympanic side there is purulent secretion with a certain definitely determined chemical property. On its labyrinthine side is a space filled with body fluids of the usual amphoteric reaction. The usual exchange, or diffusion, of chemicals through this membrane results. A sort of dialysis takes place, and as a result of a change in the very constitution of the intralabyrinthine fluid which results, cell-structures of the mebranous labyrinth become affected. Thus, it can happen that a labyrinthine reaction can be brought about, and the first phase of a change in membranous labyrinthine structures can be induced, which, according to the degree it attains, the severity of its attack, and its duration before relief is brought about, will deleteriously affect the end-organ terminating the end-filament of the distribution of the acoustic nerve. This is the result of tympanomastoidal bacterial infection. Induced intralabyrinthine disturbances are thus to be observed to occur without discernible destruction, or any loss in contiguity of the tissues of the membrane in the round-window.

Clinically, it is impossible to differentiate such an induced labyrinthitis from one which is caused by an actual break through the contiguity of the membrane. A key to the probable occurrence of a break through may be moot when one is dealing with a case of so-called acute necrotic otitis, or tympanomastoiditis (Wittmaack). For, when we know that the tympanomastoidal lesion is of the necrotic type, then it may be assumed that the labyrinthine capsule, its windows, and even the promontory, may have been the site of a necroticizing lesion.

When the ordinary diffusion and dialysising factors have continued for a time, dependent upon the intensity of the

reactions and also upon the nature of the inflammatory process, perforation of the membrane ensues, and the mass invasion of the intralabyrinthine channels are effected and is in being.

The degree to which the tissue of the two labyrinthine window membranes resist penetration or destruction—in other words, the degree of their vulnerability—depends on the variation of cell structures of the lining membrane of the middle ear spaces. Wittmaack has made a pertinent observation regarding this phase of the subject. He has described queer adhesive growths which encircle the niches of the labyrinthine windows in the presence of a hyperplastic lining membrane of the middle ear spaces. He terms them cysts whose interiors contain fluid of the nature of a transudate. From the interior of these cysts a progression of substances reach the labyrinthine interior. This is especially true of these cystic formations whose fluid pressure breaks through the neighboring membrane spanning the labyrinthine window. This has been known to happen, and an invasion of the internal ear has been brought about even in the face of a receding tympanomastoidal infection. Hence, there cannot be established definitely a relationship, clinically or pathologically, between the severity of the tympanic infection, and its tendency toward producing a perforation of the membrane spanning the labyrinthine windows.

There is another factor in mechanics of invasion to be considered. The direct action of bacteria in the tympanomastoidal region loosens the cell-strata of the layers in the tissue of the window membranes, and bacteria and their products infiltrate. In pneumatized bone, where a necroticizing lesion is developing, such a necrosis, of course, may involve a direct destruction of the membrane itself and lay open the way to a mass infection of the middle ear. Where the necrotic lesion attacks the labyrinthine capsule, the outer layer and the middle layer often succumb, leaving the endosteal layer intact. The reparative properties of these two outer layers are very slight, and the endosteal layer affords poor protection, and both diffusion, dialysis, and even penetration by bacteria, results. In the end of the sequence of events, the membranes of the labyrinthine windows break down, and mass entrance of both bacteria and their products ensues.

In the above I have briefly sketched the entrance of the products of infection into the labyrinthine channels through natural pathways. These mostly are affected in acute inflammatory reactions to what we clinically term the acute diseases of the middle ear and its adnexa. To complete the story of the mechanics of labyrinthine invasion, with its resultant destruction of acoustic nerve tissue, it is necessary to consider next the pathways created by erosions of the labyrinthine capsule, which occur mostly in the middle ear chronicities, especially in cases with cholesteatoma.

Erosions into the labyrinth, opening channels for infection, develop from the middle ear lining membrane. The breakdown of the bony structure of the labyrinthine capsule is not here analogous in its mechanics to the breakdown of intercellular bony wall structure of mastoid cells in pneumatized bone. The labyrinthine bony capsule can only break down through the operation of a process of rarefication osteitis. In the acute necrotic process the lesion first attacks the lining membrane, and Nature replaces this by an ingrowth of epithelium. The bone structure becomes absorbed as a reaction to this cholesteatomatous growth and pressure. The absorption takes place along the small blood vessel channels and spreads along Howslip's lacunae. The presence here of large numbers of osteoclasts, giant cells, are observed spreading in all directions in the bone layers, and as the process continues, a mass defect is noted. Because of the habitual site for a cholesteatomatous growth in the epitympanic space, its inflammatory reaction in the bone adjacent to it—namely, the horizontal semicircular canal—is often noted, prior to an invasion of the labyrinthine channels. The endosteal capsular layer is often spared, but sometimes it, too, is involved in the absorbing process. Occasionally the same process involves the hard bone of the Fallopian canal, absorption proceeds until the facial lies in contact with the cholesteatomatous mass, to eventually also become involved in pressure phenomena.

The process which I have just described results in what is clinically termed a labyrinthine fistula. This is a newly-formed channel whereby a tympanogenic infection often reaches the membranous labyrinth and eventuates in destruction of its nerve tissue elements. The entire labyrinth can and occasionally does become involved in the process. Often

the endosteal layer of the labyrinthine capsule alone is resistant and persists untouched by the procedure. Repair occurs by connective tissue replacement of the nerve elements and, of course, these bring with them the loss of function which the absence or destroyed nervous tissue entail.

As the fistula grows, and when only a strata of cell structure still remains to protect the intralabyrinthine fluids and membranous labyrinthine tissue in the face of an infection, for cholesteatomata often become secondarily infected, there results, then, the same diffusion of fluid contents, the same process of dialysis and similar labyrinthine reactions as have been described above as happening in and about the anatomic windows of the labyrinthine capsule, with exactly the same effect on its nerve tissue.

The causative agent of the inflammatory reactions is to be sought in the invading bacteria, or in the tissue reactions to their toxins. The intralabyrinthine tissue reaction varies from a transudate of moderate degree up to an intense exudative process. Occasionally it has been reported that an invasion has taken place, with exactly analogous phenomena from erosion of the superior semicircular canal, due to erosion of the tegmen antri from an expansion growth of a subdural abscess located over that structure.

The petrosal pyramid contributes a quota of labyrinthine invasions. A cellular pars petrosa reacts exactly like a pneumatic mastoid process. The coalescing lesion in a well pneumatized perilabyrinthine region spreads the elements of a lesion toward the dura and, unchecked, will reach the subdural space. Fortunately, we have all lately been concerned with the various petrosal lesions, for which a clinical picture has been outlined and is being better comprehended. The labyrinthine interior is reached and slight tissue reaction happens, and these transient perilabyrinthine symptoms are now understood and have both diagnostic and therapeutic significance. What should be stressed here, however, is the fact that those perilabyrinthine involvements may proceed and produce their characteristic intralabyrinthine reactions without necessarily causing a break of contiguity in the labyrinthine bony capsule tissue. Likewise, a coalescing lesion, originating in the middle ear, can follow tracts in the lining cell walls to the dural endosteal layer and lead an infection

inward along the upper and also along the posterior pyramidal walls.

The sublabyrinthine area of the pyramid is mostly involved when there has been an acute necrotic tympanomastoiditis, and the labyrinthine capsule succumbs early, and often large defects result. Of the frequent involvement of the promontory in this lesion, Wittmaack makes note. Both tubercular and syphilitic lesions often present similar labyrinthine points of entrance.

Reactions in the labyrinthine interior always follow perilabyrinthine involvement, with one exception. In uninfected pseudocholesteatomatous ingrowth, the mesial-lying lining membrane of the perilymphatic space may remain intact, and be continuous with the connective tissue layer of the cholesteatomatous matrix. Hence, there are occasionally observed odd defects in the horizontal canal, with cholesteatoma without intralabyrinthine reactions affecting the nerve tissue therein. Obviously, when this same cholesteatoma becomes either acutely or subacutely infected, breaks down, and bacterial invasion supervenes, then the process described above starts its developments, with a resultant inflammatory nerve tissue reaction in the inner ear. In this regard it must be remembered that the cholesteatomatous mass, if uninfected, substitutes itself for the tissue lost in the defect, and since the matrix is composed of high layers of pavement epithelium, it protects the labyrinthine interior against invasion of toxins and toxicities generally. Strangely enough, this matrix tissue does not bring on an antagonistic reaction between itself and the cell layers of the endosteal labyrinthine capsular layer.

The picture within the labyrinth itself in all these conditions is the same. It varies in degree of intensity, due to kind and nature of the irritant. It primarily takes the form of a *hydrops labyrinthi.* This is an outpouring of fluid, at first in the cell strata and then, as it increases in intensity and amount, into the endolymph spaces. It is somewhat analogous to the reaction which we were wont to call meningitis serosa to an acute hydrocephalus. The fluid pours out without fibrin content, and fibrin deposits are not noted. It actually is a hypersecretion. It is a similar process to the hypersecretion of the choroid plexus cells, to which I called attention, as the primary reaction to meningeal invasion by bacteria. The

factors which call it into being are not clearly understood. The suppurative or necrotic lesion in the tympanomastoid, or in the meninges first produces a serous or serofibrinous exudate in the perilymphatic space. This is an inflammatory secretion, protective in nature, and is probably designed to ward off any purulency seeking entrance to the internal ear. The endolymphatic space in turn is irritated by this secretion in close proximity to it and a hypersecretion of endolymph results. Whether this is due to heightened cell reaction of epithelial cell tissue, or whether it is an outflow of serum from dilated capillaries in the stria vascularis which bring the fluid from the intercellular stroma spaces, or whether it is a chemical ionization reaction, remains for the present an unanswered question. That it is not a direct reaction to the presence of bacteria seems established.

Hydrops labyrinthi shows itself in the endolymphatic spaces and occasionally in the perilymphatic spaces. This reaction within the labyrinth holds the key to the nerve destruction which we are today considering. It is a complication of acute tympanomastoidal infections. Its occurrence is enhanced where the tympanomastoid areas are lines with strongly hyperplastic tissue. Hence, we find it to occur in the acute exacerbations of chronicities, for chronicities of the middle ear produce this hyperplastic tissue.

If the antecedent causative factor is eradicated, this hydrops disappears as the irritant is removed, to reappear if the causative factor reappears in the case. I shall not have time to trace the changes which happen as the hydrops labyrinthi becomes further infected, to produce the well known pathology of a purulent labyrinthitis. As an intermediary stage there is often presented perilymphatic lesions with fibrinous deposits. This purulent labyrinthine involvement generally takes four forms: the tympanogenic, the meningogenic, the hemorrhagic and the necrotic form. It would take me too far afield today to outline the salient pathology of each of these types and trace their evolutionary development.

Suffice to say that the hydrops labyrinthi is the initial stage of all of them. Furthermore, from experimental data, it is evident that when promptly relieved it recedes, is absorbed and leaves little trace of its presence behind it. The various purulencies obviously cannot heal with such

results. They leave scars and tissue destruction in their wake. But this factor must be stressed in our symposium today. The duration and the intensity of the hydrops labyrinthi determine whether or not the nerve elements of the membranous labyrinth shall survive or not. If the fluid pressure within the endolymphatic space is too great, endures too long, or persists to merge into a purulency, the nerve destruction proceeds, and the delicate terminal organ of the acoustic nerve suffers damage beyond repair, even if the fluid is eventually absorbed, and the advent of purulency prevented. The nerve filaments will often be destroyed beyond repair before purulency is established clinically by the presence of the fluid itself. Hence, it is evident that the essential factors which tend to produce a hydrops labyrinthi should be better understood and, wherever possible, obviated. When the endorgan of the acoustic nerve in the membranous labyrinth is destroyed, whether by pressure exerted by the nonpurulent fluid of a hydrops or by frank purulency, there is no therapy, surgical or nonsurgical, that we know which will restore it to useful function. Those patients who survive an attack of any degree of actual severity are permanently deafened.

71 East 80th Street.

SYMPOSIUM.

THE NEURAL MECHANISM OF HEARING.

V.—ETIOLOGICAL AND CLINICAL TYPES OF SO-CALLED "NERVE DEAFNESS."

(b)—"NERVE DEAFNESS" OF KNOWN PATHOLOGY OR ETIOLOGY.

NERVE DEAFNESS FROM NONINFLAMMATORY LESIONS.*†

DR. EDMUND P. FOWLER, JR., New York.

PAGET'S DISEASE.

Paget's disease, or, more properly, "osteitis deformans," has been known for years to produce tinnitus and deafness. In a series of 99 cases from the Columbia-Presbyterian Medical Centre,[1] deafness was the initial symptom in three cases and was a major symptom in 41 cases. Tinnitus was complained of in 10 of the 99 cases and vertigo in 23. Some of these cases had marked external deformities and some had very slight deformities. The disease was shown by X-ray to be a very spotty affair. This accounts for the fact that very often the deafness was more marked in one ear than in the other, and occasionally no deafness was present at all, in spite of the fact that there was a marked deformity of the skeletal system or of the skull. Audiograms show that the typical deafness of Paget's disease is a so-called "nerve deafness." Some of the cases have a moderate or very severe loss for both air and bone conduction over the entire scale. In some cases there is simply a high tone loss. In several there is, in addition, a conduction loss, that is, there is normal bone conduction for the low notes, with a lowered air conduction for corresponding notes and then a sharp drop for both A.C. and B.C. in the high notes. In a few cases there was a marked air conduction loss, with a relatively normal

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bone conduction loss, but these patients gave a history of chronic otitis media in the affected ear.

The mechanisms producing nerve deafness in Paget's disease are quite clearly mechanical. Anyone examining the massive skull and compressed brain of the average Paget's disease, as well as the narrowing foramina for the exit of the various cranial nerves, can readily see how the disease may have a direct pressure effect upon the VIIIth nerve and cochlear vessels. Certain skulls are so deformed that the entire petrous pyramid is rotated. Indeed, I have seen one skull in which the internal auditory meati presented upwards instead of backwards. This would undoubtedly stretch the soft VIIIth nerve and compress the vessels which accompany it. Microscopic serial sections confirm the impression obtained with gross specimens. They show the narrowing of the internal auditory meatus, invasion of the modiolus and the pressing of the new bone growth on the nerve, as well as partial degeneration of the nerve trunks. In addition, they show that the bone growth invades and actually fractures the labyrinthine capsule, so as to deform the cochlea and so affect the end-organ of hearing, as well as the nerve trunk.

The studies of Alexander Gutman and his coworkers[1,2] show that osteitis deformans (Paget's) can be very definitely differentiated from most other bone diseases, including metastatic carcinoma by phosphatase determinations. He finds that the average Paget's runs a very much higher phosphatase than any other disease. Since deafness, or tinnitus, or both, have been observed in early cases without obvious bony deformities, it is suggested that phosphatase determinations be made in all cases of obscure nerve deafness. Changes in serum calcium, phosphorus and cholesterol indicate some other bone disease for these are usually normal in Paget's disease.

Radiotherapy has been found to arrest the growth of the disease enough to control the pain over the long bones, which is often a most serious symptom of this disease. This indicates that radiotherapy should be applied to the temporal bones as soon as growth near the internal auditory meatus is suspected; that is, in cases of skull involvement radiotherapy should be used even before deafness supervenes. In the one case in which we have tried it deafness with tinnitus

was already present. In this case there was a marked decrease in the tinnitus, with a consequent improvement in hearing at first, but now, three years later, the audiogram shows that the improvement has not been maintained. This single case is no argument against the use of radiotherapy for prophylactic treatment and since no other more successful treatment is available, I think that X-rays should be used in all cases in hopes of arresting, or at least slowing down the progress of the disease.

Fig. 1. Case 11942L. Labyrinthine capsule surrounded and invaded by osteitis deformans.

OTOSCLEROSIS.

Otosclerosis has been considered by some to be a localized form of Paget's disease. In an effort to check this and to see if there is enough activity in an otosclerotic focus to change the serum phosphatase, a series of phospatase studies in otosclerotics has been begun. So far, out of 14 cases, only one has shown phospatase above normal limits and this is only 4.4 Bodansky units, which is a figure just above the average normal for adults.

Even if there is no chemical similarity between otosclerosis and Paget's, there is certainly some histological rememblance.

Perhaps the most convincing evidence that the two are even different histologically is given by Otto Mayer's[3] case, in which Paget's disease and otosclerosis were present in the same individual. The lesions looked entirely different when placed side by side, and anyone studying a large series of either disease becomes less and less impressed by the similarity of the two conditions. Otosclerosis is very likely to take a heavier basophilic stain. It shows much less mosaic structure, and more important still, it always picks out the same areas to invade. As mentioned before, Paget's disease is spotty and irregular. Otosclerosis occurs almost invariably at the anterior border of the oval-window. It may occur near the round-window or the internal auditory meatus, but in these cases the foci near the oval-window are also present.

If otosclerosis invades the anterior border of the oval-window and involves the annular ligament, it produces deafness, and this deafness has been shown for years to be of the conduction type. On the other hand, certain cases of otosclerosis have been shown to have a nerve deafness for the high tones. Forty-five in a series of 126 cases reported have shown a typical conduction deafness for the low tone and have shown a sharp drop for air conduction in the highest brackets. A.C. loss for 8,192 and 4,096 has been interpreted as a nerve deafness without any bone conduction readings for these frequencies; this is an erroneous conclusion, as is shown by audiograms in which there is a normal bone conduction throughout the scale and a moderate air conduction loss for the high tones. On the other hand, there are certainly cases of otosclerosis which have a more or less severe loss of bone conduction for individual notes; this is usually interpreted as a nerve deafness. A very few of these can be explained by otosclerotic foci which grow in the region of the internal auditory meatus and press on the VIIIth nerve trunk or deform the lower turn of the cochlea or the modiolus. The remainder of the cases of otosclerosis showing so-called nerve deafness are more difficult to explain. The fact that they are more often found in older people and are usually present for the higher tones suggests that many of these cases, if not all of them, are simply the high tone loss which is so commonly found in older people, whether they are conscious of it or have a history of ear disease or not. The average variations in bone conduction throughout the decades,

reported before this Society last year, show how great the bone conduction losses can be without the patient and, occasionally, the otologist being aware of it.[5]

It is theoretically possible for bone conduction losses to be due to closure of the round-windows,[6] for the endolymph and perilymphatic vibrations would be dampened if there were no soft tissue release for them; however, this type of case would be interpreted as a nerve deafness. We have

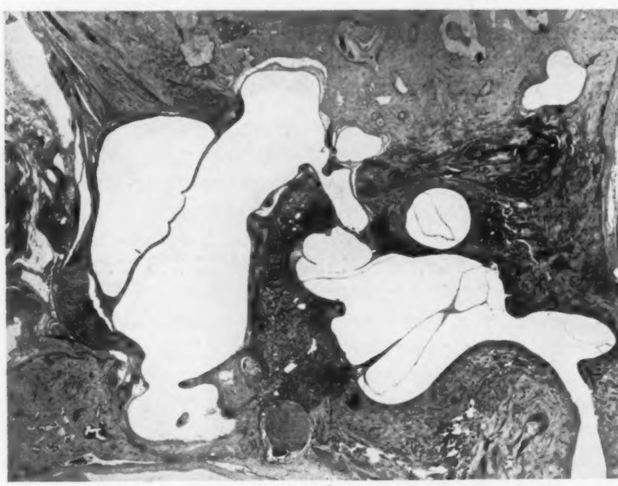

Fig. 2. Case 11090R. Case of otosclerosis showing partial blockage of the round window by the growth. This case had a shortened bone conduction which theoretically could be due to blockage of the round window.

in our collection such a case. There was a marked decrease for the 128 and 256 forks for both air and bone conduction on one side. This side showed partial obliteration of the round-window by a large otosclerotic focus. Unfortunately, the histological preservation in this case was not good enough to be sure that there was not in addition some end-organ atrophy, but the spiral ganglion was intact, so that if an end-organ lesion was present, it must have been minimal.

A careful study of the VIIIth nerve of otosclerosis in our collection and 14 of otosclerosis presented to the research

council of the American Otological Society by various laboratories abroad fails to reveal convincing proof that the conclusions reached by Albert Gray[7] concerning the histological pictures found by him in the VIIIth nerve are correct.

There is, therefore, no convincing proof either to be found in actual sections or in the literature that nerve deafness in otosclerosis is anything different than the nerve deafness so often found in a cross-section of the population at all ages, and especially in people over 40 years. No nerve deafness is present in many individuals who have been known to have been hard of hearing for years; so that the old story of atrophy due to disuse is also open to question.

TUMORS.

Various types of benign as well as malignant growths may produce nerve deafness. Exostoses of the internal auditory meatus have been described and are occasionally thought to press on the nerve. The etiology of these exostoses is obscure. Epitheliomata of the external canal and the middle ear occasionally grow so large as to invade the labyrinth and to produce nerve deafness. Likewise, primary carcinomata and sarcomata, as well as metastatic carcinomata, may invade the temporal bone until they involve the labyrinth and so produce nerve deafness. More common than these are cholesteatomata. These are usually the pseudocholesteatoma type; that is, the ingrowth of the squamous epithelium of the external canal which grows in the attic and curls up there into such a large ball that pressure and corrosion results. With all types of growth there is usually a labyrinthitis which produces a dead labyrinth.

We have in our collection one case of a granulomatous lesion in the VIIIth nerve due to long standing meningitis. Lymphatic invasion of the VIIIth nerve and labyrinth has been described, but we have not been fortunate enough to obtain one in our collection. Of 25 cases of lymphatic and myeloid leukemia, we have not a single one which shows a curve suggesting leukocytic invasion of the labyrinth or VIIIth nerve, and in five cases which have been serially sectioned none show invasion of either labyrinth or nerve.

A very special type of nerve tumor develops more commonly from the VIIIth nerve than from any other nerve in

the body. It has been given various names, including neurilemoma, neurofibromia, Schwannomma, etc. These grow into the classical cerebellar pontine angle tumors and until the histologist definitely decides their origin, are perhaps best called acoustic tumors.[8] These types of tumors of the VIIIth nerve have been described, the most striking being neurofibromatosis (so-called Von Recklinghausen disease), described by Felix Nager and others.

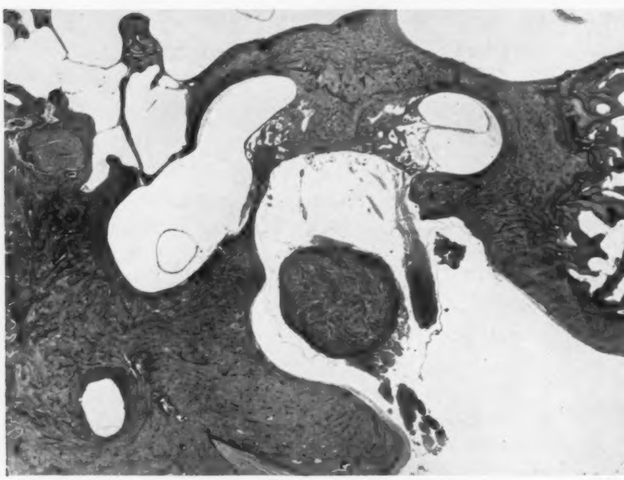

Fig. 3. Case 11009L. Small acoustic tumor within the internal auditory meatus, showing region where cerebellopontine angle tumors usually originate.

Hardy and Crowe described a most unusual tumor, which looked like misplaced glial tissue.[9]

Fractures through the temporal bone often involve the labyrinth. If they do, hemorrhage into the vestibule and cochlea is the rule, and a severe nerve deafness usually results.[7] In fact, the deafness is usually total.

In the newborn, deafness may be produced by rupture of vessels or nerves in the labyrinth or in the internal auditory meatus by deforming of the head of the fetus as it passes through the birth canal.

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SYMPOSIUM.

THE NEURAL MECHANISM OF HEARING.

V.—ETIOLOGICAL AND CLINICAL TYPES OF SO-CALLED "NERVE DEAFNESS."

(b)—"NERVE DEAFNESS" OF KNOWN PATHOLOGY OR ETIOLOGY.

FROM CENTRAL OR CORTICAL LESIONS.

(a)—PARTIAL SECTION OF THE VIIITH NERVE.*

DR. WALTER E. DANDY, Baltimore.

From a series of human intracranial operations, which serve as experiments, I wish to bring you certain facts concerning the effects upon hearing in two different lines in the pathways of hearing.

This sketch is that which you will see in any textbook, with the exception of the central pathways for hearing; *i.e.*, those in the cerebral hemispheres.

It is, I think, universally claimed that the centre for hearing lies in each temporal lobe. How this conception has arisen is difficult to understand, but it is certainly a mistake, for one can remove either the entire right or left temporal lobe with no effects whatsoever upon hearing. Audiometer tests have, of course, been used in checking these experiments.

Another more extensive experiment is perhaps even more impressive when the entire right cerebral hemisphere is removed—this has been done seven or eight times—there is not the slightest effect upon hearing.

After removing the occipital, frontal and temporal lobes on the left side (in different individuals), there is no effect upon hearing; therefore, the only possible location for a centre of hearing is in the left parietal lobe. It is unquestionably connected with the auditory speech centre, which

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is located in the parietal lobe, and not in the temporal lobe, as most textbooks state.

In this connection, two interesting points from a negative standpoint are: *1.* that between the mesial geniculate body and the inferior colliculus anteriorly, and the point of entrance of the VIIIth nerve into the brain stem posteriorly, I have never seen a brain tumor that has produced deafness; and, *2.* I have never seen a tumor in the cerebral hemispheres, right or left, that has produced deafness unless there has been an associated deficiency of auditory speech. These facts must carry significance, although I am not prepared to make

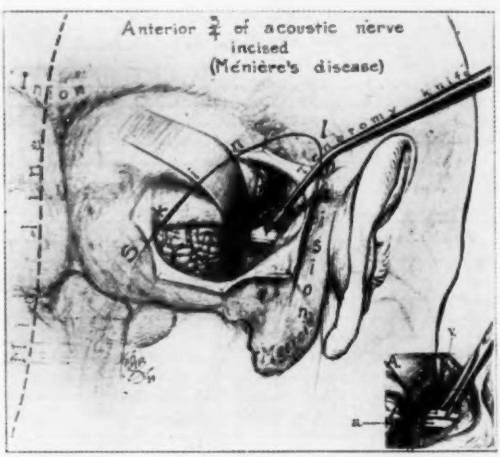

Fig. 1. Method of fractional division of the auditory nerve. The anterior half of the auditory nerve is the vestibular division; the posterior half, the auditory division.

a positive explanation. Possibly there may be one central region in which defects of hearing may carry diagnostic significance; *i.e.*, at the inferior colliculus or the medial geniculate body. I have had three cases in which tumors in this region (pineal or adjacent tumors) have shown an abrupt bilateral drop in the high tones. These may have been purely coincidental findings, but I believe they are significant. In one case where an audiometer test had not been made, the tumor was found in this location, a subsequent check-up disclosed this same suspected finding.

Other than this, when deafness results from an intracranial tumor the lesion is always located along the VIIIth nerve itself. It is, therefore, of the greatest and most certain diagnostic import.

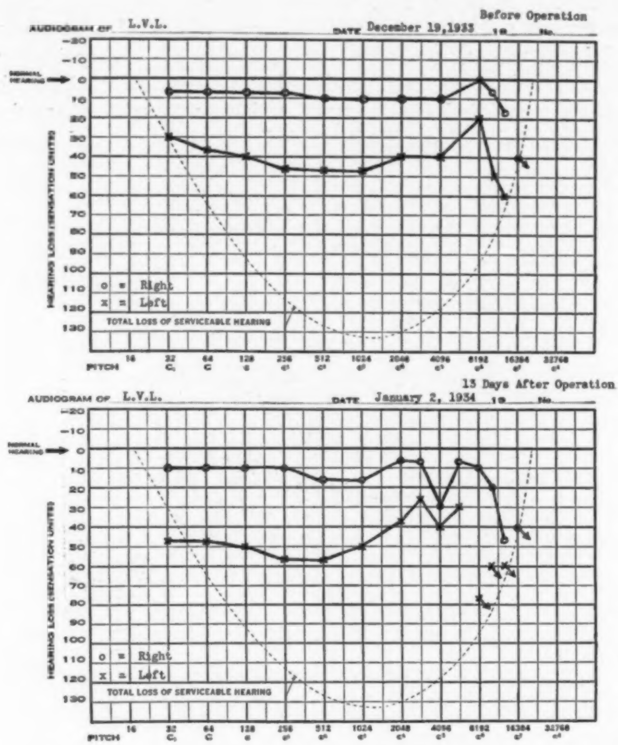

Fig. 2. Audiograms before and after division of three-fourths of the auditory nerve, or one-half of the hearing division of the nerve. The only loss of function is in the very high tones.

Why the absence of defects of hearing from tumors throughout the brain stem? My only explanation is that the fibres scatter after the arrival in the brain stem.

My next point of experimental attack is in the VIIIth nerve itself; *i.e.*, in the intracranial portion between the spiral

ganglion and the brain stem. From a large series of operations for Méniére's disease (Fig. 1), we have found this one very interesting observation; namely, that one can divide one-half to two-thirds, at least, of the auditory division of the VIIIth nerve and lose practically nothing in hearing (Fig. 2). When slight losses do occur they are restricted to the very high tones.

In Méniére's disease, where deafness is always associated with the attacks, the lesion must be in the nerve itself. In several specimens removed at operation there has been fibrosis of the nerve. This is, I think, the explanation of the syndrome. It is the only intrinsic change I have been able to find. In about 20 per cent of the cases a large artery (one of the branches of the superior-inferior cerebellar artery) lies against the nerve, and is, I think, one of the causes of the deafness and dizziness of this disease.

SYMPOSIUM.

THE NEURAL MECHANISM OF HEARING.

V.—ETIOLOGICAL AND CLINICAL TYPES OF SO-CALLED "NERVE DEAFNESS."

(b)—"NERVE DEAFNESS" OF KNOWN PATHOLOGY OR ETIOLOGY.

FROM CENTRAL OR CORTICAL LESIONS.

(b)—DEAFNESS DUE TO BRAIN TUMORS, ANGLE TUMORS, VESSEL ABNORMALITIES, OTHER CENTRAL AND CORTICAL LESIONS.*

DR. HAROLD G. TOBEY, Boston.

Since this discussion has to do with deafness caused by brain tumors, cerebellopontine angle tumors and vessel anomalies, it seems best to reverse the order and pass from the peripheral to the central lesions and from the known to the relatively unknown.

Vessel Anomalies: Dandy has shown, from his experience in the surgical relief of Méniére's disease, that vessel anomalies may not infrequently be the cause of the vertigo and deafness. From a comparatively short series of cases, he cites the occurrence of two tumors and one aneurysm of the basilar artery which seem to have been the cause of the symptoms, and a series of five large arterial loops (from the anterior inferior cerebellar artery) in the lateral cistern which were equally positive lesions in the production of the disease. He also suggests that vessel anomalies in more inaccessible situations may be a possible cause of symptoms. It is presumed that with the early recognition and correction of the condition, no permanent deafness will result.

Cerebellopontine Angle Tumors, as is well known, are a frequent cause of deafness. Dandy again reminds us that the early symptoms of these tumors are the symptoms of Méniére's disease. It is customary, in the presence of a suspected pon-

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tine angle tumor, to wait for further symptoms, such as papilloedema, headache and facial paralysis, before surgical interference is undertaken. It may well be that, in the light of recent experiences in the surgical treatment of Méniére's disease, tumors of the cerebellopontine angle may be approached at an earlier stage. This is important, for Crowe has shown that the deafness resulting from the pressure of the tumor is not only due to a localized degeneration of the auditory nerves, but that the degeneration involves the peripheral nerve fibres extending even into the spiral ganglion and the organ of Corti. Therefore, the deafness resulting from cerebellopontine angle tumors is that of perception.

Brain Tumors: We now enter upon less well known and debatable ground. It may be well to state at the beginning that the consensus of opinion, as reported in the recent literature, is against the assumption of brain tumors, aside from the pontine angle tumors, being an important cause of deafness.

Kolodny, 1928, found three cases of deafness in a series of 38 brain tumors; in each of these the tumor was of the temporal lobe.

Drury, 1931, in a series of 361 cases of brain tumors, found that, aside from the angle tumors, only 0.03 per cent showed loss of hearing.

Frazier and Rowe, 1934, found that auditory symptoms were the most frequent sign of temporal lobe dysfunction, namely, 33 per cent. At the same time they quote Gibbs, who in 1,545 cases found that only 3 per cent showed loss of hearing, again excluding angle tumors.

Northington, 1936, reported that considerably impaired auditory function is not usual in a group of intracranial tumors.

H. Brunner lays down several general principles: first, "in cases with chronic generalized brain pressure, but without localized pressure on the brain stem, the hearing stays normal; second, in cases with chronic localized pressure on the brain stem we have deafness occurring late in the disease, is combined with hypofunction of the labyrinth, shows a rapid progress, appears very late, and is combined with pontine

and medullary symptoms." Inferentially, we must presume that chronic localized pressure in the cerebrum does not cause deafness.

We must, therefore, differentiate between infra- and supratentorial chronic localized pressure and, in the former, disturbances of the vestibular system are the earliest symptoms.

We have, then, established deafness as caused by vessel anomalies, cerebellopontine angle tumors and brain stem tumors; and although the majority of these instances show a unilateral involvement, it may, nevertheless, be bilateral.

There remains to be considered the condition of true central deafness, to which the obstacles of a definite demonstration seem to be well-nigh insuperable.

We have seen that what evidence there is of deafness caused by cerebral tumors points to the temporal lobe as the chief site. The occurrence of the various types of aphasia in lesions of the temporal lobe is also significant.

The anatomical structure and pathways of the auditory tracts have been well worked out and seem to be conclusive, the auditory centre being placed in the superior temporal convolution and, particularly, in the transverse gyri of Heschl.

The physiological and experimental evidence as to the exact location of the auditory centre is beset with contradictions. Early experimenters often obtained exactly opposite results. At the present moment it may fairly be said that experimental evidence of the exact location of the auditory centre is lacking.

Larionow, in 1898, attempted auditory localization by obtaining galvinometric measurements of the current in the cortex of the temporal lobes during stimulation of the peripheral organs with tuning forks. This attempt was defeated by an unpreventable diffusion of the current, apparently along association tracts. This experiment foreshadowed the more recent advances in experimentation by means of the phenomenon of Wever and Bray, which, in turn, has so far been unable to definitely localize the auditory centre.

Present knowledge may be succinctly presented by the results of the experiments of Mettler and his co-workers under a grant from the research fund of this Society.

1. Removal of the cortex of a single hemisphere is followed by a drop in auditory acuity (1,000 cyc. of 2-5 dcb.).

2. In a dog, at least, there is no appreciable difference between the right and left hemispheres.

3. The removal of the entire cortex produces an enormous reduction, 70-75 dcb. below normal.

4. In animals with the cortex of one hemisphere removed, if the crossed fibres of the opposite lateral lemniscus be rendered nonfunctional, an additional drop in acuity of about 10 dcb. is observed.

5. If the uncrossed fibres of the opposite lateral lemniscus are rendered nonfunctional, an additional drop in acuity of about 10 dcb. is noted.

6. It follows that the uncrossed fibres of the auditory system are approximately equal in acoustic value to the crossed components.

7. Destruction of one cochlea in an otherwise intact animal is followed by a loss of about 3 dcb. Destruction of both cochleae produces total deafness.

8. The physiological "safety factor" in the auditory system thus seems to compare favorably with that observed in other organs of the body.

The clinicopathological evidence of true central deafness is very meagre. Campbell was able to collect five cases of total deafness established on an anatomical basis by a careful examination of the brain after death. In none of these was a brain tumor present. In one, Friedlander and Wernick, a gummatous lesion was present in both temporal lobes; Pick, a bilateral destructive lesion; Sérieux and Mignot, hydatid cysts in both temporal lobes; Anton, bilateral patches of softening; Mills, an old hemorrhagic process.

Fraser and Nelson have reported a case of a deafmute in whom the cochleae were normal and intact, but noteworthy changes were found in the cortex of Heschl's gyri.

The ideal conditions for the clinicopathological proofs of true central deafness require such a happy conjunction of circumstances as to seem well-nigh hopeless.

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270 Commonwealth Avenue.

SYMPOSIUM.

THE NEURAL MECHANISM OF HEARING.

V.—ETIOLOGICAL AND CLINICAL TYPES OF SO-CALLED "NERVE DEAFNESS."

(b)—"NERVE DEAFNESS" OF KNOWN PATHOLOGY OR ETIOLOGY.

HEARING AIDS FOR "NERVE DEAFNESS."*

JOHN C. STEINBERG, Ph.D., New York.

Recent studies have shown that the hearing for sounds of threshold intensity may not indicate how the deafened ear hears sounds of above threshold intensity.[1] The purpose of this paper is to review these studies very briefly and discuss their bearing on hearing aids.

In general, two types of deafness have been observed: In one kind, above threshold sounds are heard with less than normal loudness by the amount of the audiogram. In other words, the deafened ear hears with diminished loudness at all intensity levels of sound. In the other kind, the deafened ear hears with practically normal loudness, sounds which are 30 or more dcb. above the deafened threshold. These two kinds are called constant and variable types of deafness, respectively, because of their behavior with change of intensity level.

Fig. 1 shows results obtained at Bell Telephone Laboratories[2] which are typical of the constant type deafness. The left and right ear audiograms shown in the upper charts indicate that the left ear is normal and that the right ear has a hearing loss of about 35 dcb. The three lower charts show the decibels needed on each ear for single frequency tones to sound equally loud. They show that the deafened ear needed 20 to 35 dcb. more level than the normal ear, or the amount indicated by the audiogram.

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Fig. 2 shows results typical of the variable type deafness. Fig. 2A is one of our cases; Fig. 2B was taken from Dr. Fowler's paper, previously cited. The impaired ear in both cases shows a 60 dcb. hearing loss at 2,000 cycles. As shown in the right hand charts, a 2,000 cycle tone level of 65 dcb. on the impaired ear is equal in loudness to a 5 dcb. level on the normal ear. The impaired ear needs 60 dcb. greater tone level than the normal ear for equality of loudness, when the tone is only slightly above the deafened threshold. When

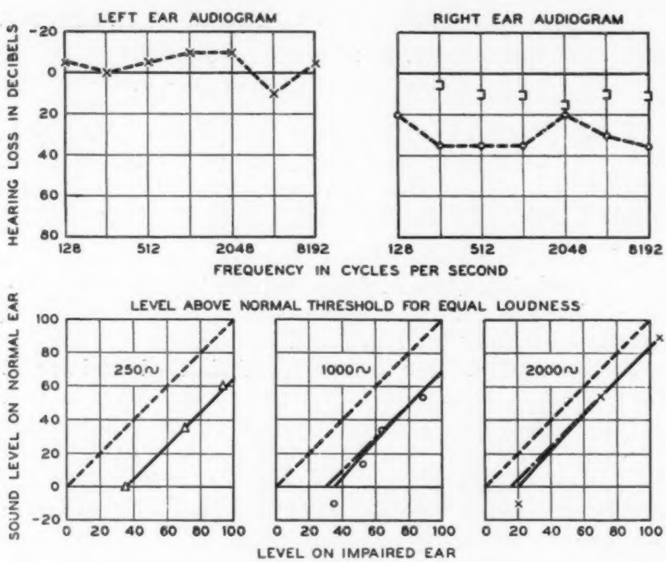

Fig. 1.

the tone is 30 to 40 dcb. above the deafened threshold it is heard with practically the same loudness on the deafened as on the normal ear. For example, the lower right hand chart shows that a tone having a level of 100 dcb. sounds as loud on the impaired as on the normal ear. For this level the impaired ear shows no measurable loss in hearing for a 2,000 cycle tone, although there is present a 60 dcb. loss for a faint 2,000 cycle tone.

In addition to the two types just described, cases were studied which were mixtures of the variable and constant

type deafness. The occurrence of these two types of deafness can be explained from our present picture of loudness. A great deal of evidence has been obtained which is consistent with the concept that the loudness of a sound bears a close relation to the total number of nerve fibres conducting impulses due to the stimulation of the sound.[3] The normal hearing curve of Fig. 3 shows the relation between the loudness for a normal ear and the intensity level of a 1,000 cycle tone. The loudness which is expressed on the y-axis on an

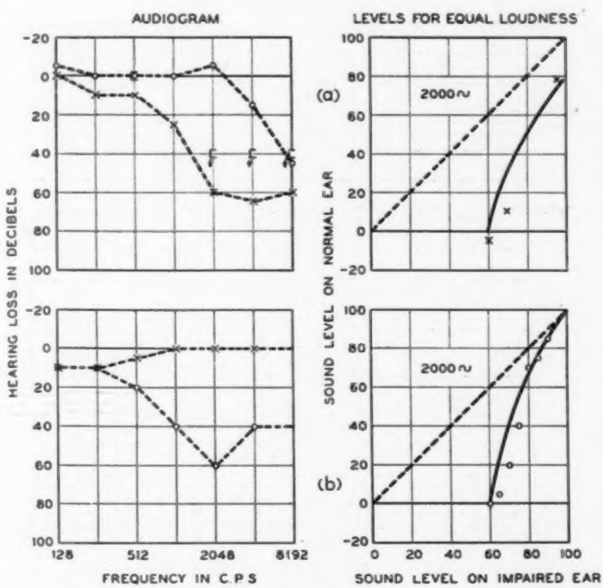

Fig. 2.

arbitrary number scale is a measure of the sensory magnitude and is thought to be closely related to the number of nerve impulses conducted to the brain due to the stimulation of the tone. It is a scale such that a tone having a loudness 2N is twice as loud as one having a loudness N. On the X-axis, the physical magnitude of the sound is expressed.

Let us take conductive deafness to mean that the physical magnitude of the sound reaching the inner ear is reduced.

On this assumption, the conductive deafened curve of Fig. 3 shows the loudness heard by an ear having a conductive deafness of 40 dcb. It was obtained by shifting the normal curve 40 dcb. to the right and shows that the loudness heard by the deafened ear is 40 dcb. less than normal at all intensity levels.

Now let us take nerve deafness to mean a loss in the number of nerve fibres that normally respond to a tone. Since the number of conducting fibres is closely related to loudness, a nerve fibre loss is equivalent to a loudness loss. On this assumption, the nerve deafened curve of Fig. 3 shows the loudness heard by an ear having a nerve deafness of 40 dcb. It was obtained by subtracting 1,000 loudness units from the normal curve. In this case, the deafened ear tends to hear loud sounds normally, as the loss of 1,000 units does not affect the loudness appreciably when a large number of units is involved.

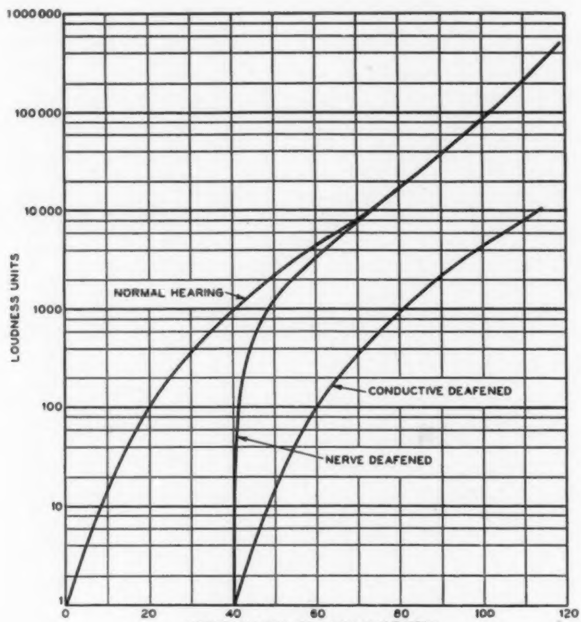

Fig. 3.

Using loudness relations based on normal ears, the loudness of tones heard by nerve deafened ears may be calculated from the audiograms.[2] For this purpose, pure tone loudness patterns for normal ears are used. These patterns are obtained from the masking audiograms of the sounds, or audiograms which show the threshold shift for single frequency tones when heard in the presence of the sound.

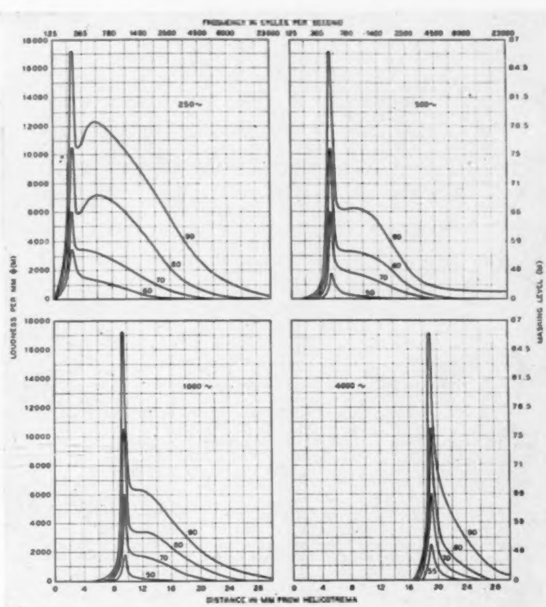

Fig. 4.

Loudness patterns for different pure tones are shown in Fig. 4. The x-axis gives distance on the basilar membrane and the y-axis gives the loudness contributed per unit length of membrane. It is proportional to the number of nerves stimulated in each portion of the membrane by the tones. The different curves correspond to different levels of tone. The scales on the upper and right hand margins of the charts show the frequency and masking values which were used to obtain the loudness patterns. The areas under the curves give the loudness as sensed by a normal hearing person. It

will be seen that an appreciable portion of the basilar membrane contributes to the loudness of a pure tone at the higher levels.

For a nerve deafened person only part of the area is effective; namely, that part which projects above the deafened threshold. The solid lines of Fig. 5 show the normal loudness areas for a 2,000 cycle tone. The dashed lines through

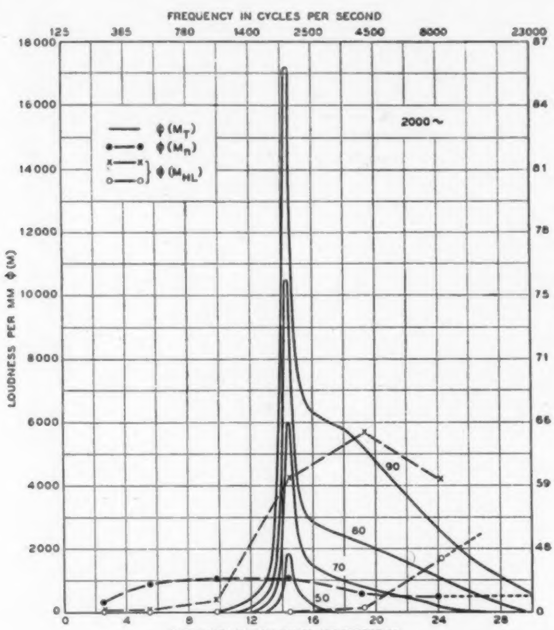

Fig. 5.

the points indicated by crosses and circles show the deafened thresholds for the left and right ear audiograms of Fig. 2A. The areas above the threshold lines give the loudness of the 2,000 cycle tone as heard by the deafened ears.

The solid line in the right hand chart of Fig. 2A shows the calculated levels for equal loudness. The crosses are observed points. It is of interest to compare the observed and calculated results in Figs. 2A and 2B. Both audiograms show a 60 dcb. hearing loss for a 2,000 cycle tone. When

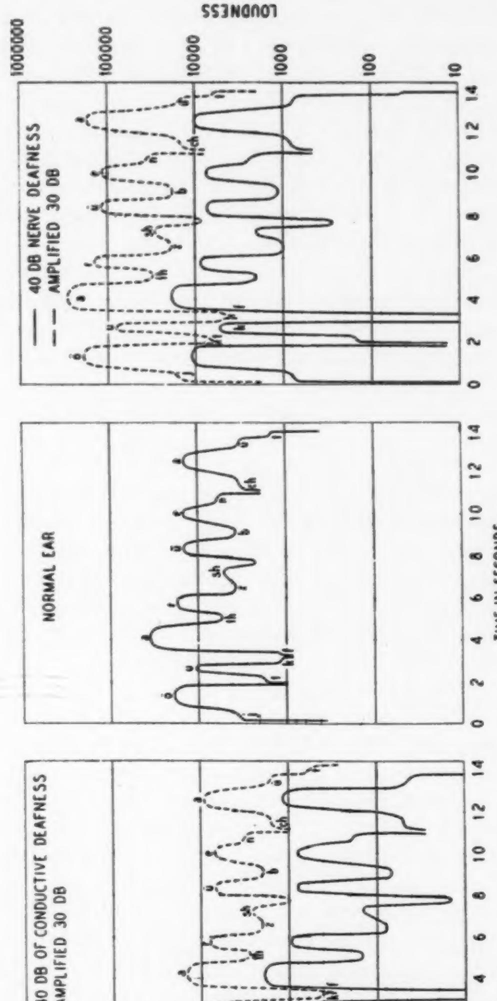
40 DB OF CONDUCTIVE DEAFNESS
AMPLIFIED 30 DB
NORMAL EAR
40 DB NERVE DEAFNESS
AMPLIFIED 30 DB
LOUDNESS
1000000
100000
10000
1000
100
10
0 2 4 6 8 10 12 14
TIME IN SECONDS

Fig. 6.

the level of the 2,000 cycle tone is raised 30 dcb. above the deafened threshold, the ear in Fig. 2B hears with normal loudness, but that in Fig. 2A hears less loudly than normally, by about 25 dcb. The difference is due to the greater hearing loss at 4,000 and 8,000 cycles in the case of Fig. 2A. Due to this greater loss, these regions contribute less to the loudness of the 2,000 cycle tone than is the case in Fig. 2B. That the calculations appraised this difference correctly gives some confidence in the method.

In view of the differences between nerve and conductive deafness that have been discussed it would be expected that the amplification of sound would produce quite different effects for the two types of deafness. In the case of conductive deafness, since it is equivalent to an attenuation, the hearing loss can practically be restored for moderate amounts of deafness by amplification equal in amount to the attenuation. Even for very great deafness, good results may be obtained with large amplifications. Frequently better results are obtained with bone-conduction than with air-conduction receivers when the levels are such as to produce feeling in the middle-ear. For this type of deafness, it would be expected that amplification which compensated for differences in hearing loss at different frequencies would be better, in general, than uniform amplification at all frequencies.

In the case of nerve deafness, loud sounds are heard with practically normal loudness, but faint sounds are heard with a less than normal loudness corresponding to the hearing loss. Any amplification to restore faint sounds to normal loudness causes loud sounds to be above normal in loudness. If sounds of a fixed loudness are being listened to, the amplification can be adjusted to give a satisfactory loudness. For variable sounds, the nerve deafened ear may be subjected to uncomfortably loud sounds when the amplification is set for restoring the faint sounds to normal.

In Fig. 6, the effects of amplification for the two types of deafness is shown for the sounds in the sentence. "Joe took father's shoe bench out." The y-axis represents loudness and the x-axis represents time, so that the graphs show the loudness variation with time as the sounds in the sentence are spoken. The middle chart shows the loudness variations as heard by a normal ear, when a speaker is 1 meter away from the listener. The faintest sounds, such as k and f, have

a loudness of about 1,000 units, and the loudest sound, a, has a loudness of 32 times as great.

On this scale of loudness, the faintest sound that can be heard has a loudness of one and the loudest sound that can be tolerated has a loudness of 1,000,000.

The solid line on the left hand chart shows the loudness variations as heard by an ear having 40 dcb. of conductive deafness. Both the loud and faint sounds of speech are greatly reduced in loudness and the faint sounds are barely audible. The dashed line shows the loudness variations for such an ear after the sound has been amplified 30 dcb. Comparison with the normal ear curve shows that the hearing of the deafened ear has been practically restored.

The solid line of the right hand chart shows the loudness variations of an ear having 40 dcb. of nerve deafness. Comparison with the normal ear curve shows that the loss for the loud sounds is not very great, but the faint sounds are near the threshold of hearing. The dashed line shows the loudness heard by such an ear after the sound has been amplified 30 dcb. The faint sounds have been restored to normal, but the loud sounds are far above the range of normal loudness and are in the range of uncomfortably loud sounds.

These curves explain the frequent comment by nerve deafened individuals, on the tendency of amplified speech to become annoyingly loud when the amplification is great enough for the faint speech sounds to be heard. When someone shouts, the amplified speech causes practically as much discomfort to the nerve deafened as to the normal hearing person under the same circumstances. This characteristic could be helped by the use of selective amplification such that greater amplification would be provided for faint than for loud sounds.

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MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

SECTION ON OTOLARYNGOLOGY.

Meeting of April 9, 1937.

Dr. Charles E. Gurney, Fellow in Oral and Plastic Surgery, The Mayo Foundation (by invitation), read a paper on "Studies on the Fate of Free Transplants of Fat." The first attempt at the transplantation of fat is accredited to Neuber, who reported the successful use of small free transplants about the orbit in 1893. Although it has been used for a variety of conditions since that time, the fat graft now finds its greatest field of usefulness in plastic and reconstructive operations. Here it is used to obliterate dead spaces, to fill small depressions in the soft tissues for the purpose of restoring the contour and mobility, to elevate depressed and adherent scars and to prevent subsequent adhesions and contractures.

In spite of its various uses, there are certain clinical problems associated with the fat graft which warrant further consideration. Why does nearly every fat transplant diminish in size? Why do some become much smaller than others? Why do some grafts undergo partial or complete liquefaction without any apparent infection? Should one use multiple small pieces rather than a single large piece of equal bulk? Does fat from one part of the body survive better than from another part? Can fat be transplanted successfully from one individual to another? Does the transplanted fat remain as fat or does it become scar tissue? Is the graft replaced by new fatty tissue formed by the host? These are practical and pertinent questions and seem to justify further investigation.

The present study consisted of autotransplantation and homotransplantation of fat in 185 male and female white rats of average size. In each instance fat was transferred from the right groin, or peritoneal cavity, to a subcutaneous pocket in the left ventral thoracic region. For all operations ether anesthesia and aseptic technique were employed. The rats were divided into five groups: In Group 1, autotransplantation of fat from the groin was performed without trauma; in Group 2, autotransplantation of fat from the groin, which was cut into multiple small pieces, was performed; in Group 3, autotransplantation of fat from the groin, which had been severely crushed immediately before transplanting, was done; in Group 4, homotransplantation of a single piece of fat from the groin was performed without trauma; and in Group 5, autotransplantation of a piece of fat from the peritoneal cavity, which was entirely surrounded with peritoneum except for a small portion where it was attached to the testis and epididymis.

All rats were killed at intervals of one, two and three weeks, and one, two, three, four, six, nine and 12 months, except those of Group 5, which were killed at intervals of one week and one, four, nine and 12 months. Each surviving graft was measured for purposes of comparison of its size with its original size. It was sectioned later and stained with sudan III or hematoxylin and eosin for microscopic study.

Results: Almost without exception, the autografts which were not traumatized retained their identity throughout the whole year of observation. At the end of the year, the transplants of fat from the groin were about a fourth their original size, whereas those of testicular fat were about half their original size. The autografts which were cut into multiple pieces and those which were subjected to severe trauma had begun to disappear at the end of two months and, with one exception, were entirely gone at the end of six months. The homografts conformed to the accepted opinion regard-

ing homografts in general, for they had disappeared entirely by the third month. Gross infections resulted in the complete disintegration of the transplant in seven rats. With one exception, all cases of infection occurred in in the group in which grafts had been subjected to cutting or crushing. In several instances in which evidence of a hematoma was found, the graft partially or completely degenerated.

The findings on microscopic examination revealed that certain portions of the grafts which survived were becoming vascularized as early as one week after transplanation. Other portions of the graft degenerated and disappeared, leaving only the normal appearing fat. The histiocyte seemed to be the chief phagocytic cell for the fat, which was liberated by the degenerating fat cells. No giant cells were found in any of the sections. A futile search was made to find new fat cells, which are thought by some workers to be formed by the tissues of the host in response to the degenerating graft. In those instances in which the graft had disappeared completely, an effort was made to find scar tissue in its place, but it was not to be found. The remaining tissue of those grafts which did survive was not scar tissue, but normal appearing fat. Apparently the reduction in size of the transplants was attributable to degeneration of a portion of the graft, and this in turn was related definitely to the amount of trauma to which the graft was subjected prior to transplantation. There was no complete liquefaction in any of the grafts. The nearest approach to it was an unusually large amount of oily fluid, which escaped from the cut surface of those grafts which had been cut or crushed. This observation suggests that trauma might be a factor in its production.

Conclusions: The following conclusions seem to be justified:

1. The transplantation of multiple pieces of fat, in lieu of a single piece of equal bulk, is not a satisfactory surgical procedure.

2. The fat which is found in the graft one year after transplantation is a portion of the original fat.

3. A much larger piece of fat must be transplanted than would seem necessary at first, because only from a fourth to a half of the graft survives.

4. Trauma is apparently a definite factor in the reduction in size of the graft.

5. The original fat which is removed for transplantation is not replaced during the first year after such removal.

DISCUSSION.

Dr. K. C. Wold asked Dr. Gurney if he could draw the same conclusions with regard to human implants and would the same percentages hold true?

Dr. Gurney said the same question might arise with regard to transplantation of other tissues. It is logical to assume that the changes would occur in the human as well as in the experimental animal. It is true for cartilage and bone, and there is no good reason why it would not hold true for fat.

Dr. E. J. Borgeson asked what is the difference in shrinkage between cartilage and fat?

Dr. Gurney said that cartilage does not shrink. Cartilage is cut the same size as it is expected to be ultimately, whereas fat always shrinks.

Dr. F. Z. Havens stated that he had had occasion to use and to observe the use of fat to correct depression deformities in a number of instances. Before this experimental work was done, it had been his custom to insert enough fat to provide about 50 per cent overcorrection and the final result in these cases had been quite satisfactory. Whether there is not as much shrinkage in transplanted fat in the human as in the experimental animal, he did not know.

Dr. Borgeson asked Dr. Gurney if he knew the difference in the amount of shrinkage between one to 10 years.

Dr. Gurney replied there is good reason to believe that a fat transplant shrinks very little, if any, after one year. At that time the remaining fat is well vascularized and the degenerated portion has been removed from the implant site. It has the appearance of normal fat.

Dr. H. I. Lillie read a paper on "Mandibular Fossa Abscess Secondary to Otitis Media and Mastoiditis."

The author gave a preliminary report of two cases in which an abscess in the mandibular fossa had caused pain and swelling in front of the ear, and severe trismus. In the first case, an exploratory operation revealed an acute abscess. An abscess in the zygomatic fossa had been expected. In the second case the abscess was chronic and the surgical interference was purposely directed to the mandibular fossa. The extension of the infection from the ear is believed to have taken place through the glaserian fissure. From the mandibular fossa the abscess may extend to the pterygomaxillary fossa. Direct surgical approach to the mandibular fossa, the site of origin of the abscess, was emphasized as being preferable to external drainage of the secondary extension of the abscess because, if extensive disease is encountered in the mandibular fossa, as it was in the chronic abscess, it must be dealt with in an adequate manner. The details of the exposure of the mandibular fossa were outlined.

Dr. Fred Z. Havens read a paper on "Primary Melanoepithelioma of the Nasal Mucosa."

It is generally conceded that the malignant melanotic tumors constitute the most vicious group with which we have to deal, chiefly because they frequently give rise to early and widespread metastasis.

A study of the literature dealing with melanoepithelioma of the nose was prompted by the observation of two patients with melanotic malignant tumors arising from the nasal mucosa, who came almost simultaneously to the Mayo Clinic in 1930. Reports of 28 cases were found. Seven of the patients were said to have been free from recurrence following treatment for periods varying from six and one-half months to 15 years, with an average period of 37 months. All of the remaining patients died of their disease. The method of treatment in all of the cases reported free from recurrence had consisted of some type of removal, either by cautery excision, by wide resection of the involved area, or with surgical diathermy.

The outstanding early symptoms are nasal obstruction and a bloody discharge. The diagnosis should be strongly suspected on the finding of a black or purplish tumor in the nasal cavity. Occasionally polypi are seen which have such an appearance on account of hemorrhage having occurred within them.

Surgical diathermy plus irradiation was used for the treatment of the two cases observed in 1930. One patient lived comfortably for four and one-half years, but eventually died with extensive recurrence. The other patient lived comfortably for 21 months, but died at the end of three additional months, with extensive generalized metastasis. These two cases, together with one hopeless case previously seen, make a total of 31 cases reported (as of April 1, 1933).

In the presence of a nasal lesion that may prove to be a malignant melanotic tumor, with its attendant high mortarity (77.5 per cent), it is imperative that one proceed with utmost caution if the patient's best interests are to be served. The writer believes it to be bad practice to remove all or part of such a tumor for examination unless one is prepared to secure an immediate microscopic diagnosis and to proceed at the same sitting with radical removal or wide destruction of the tumor. Surgical diathermy is, in the author's opinion, the best method for removing these lesions.